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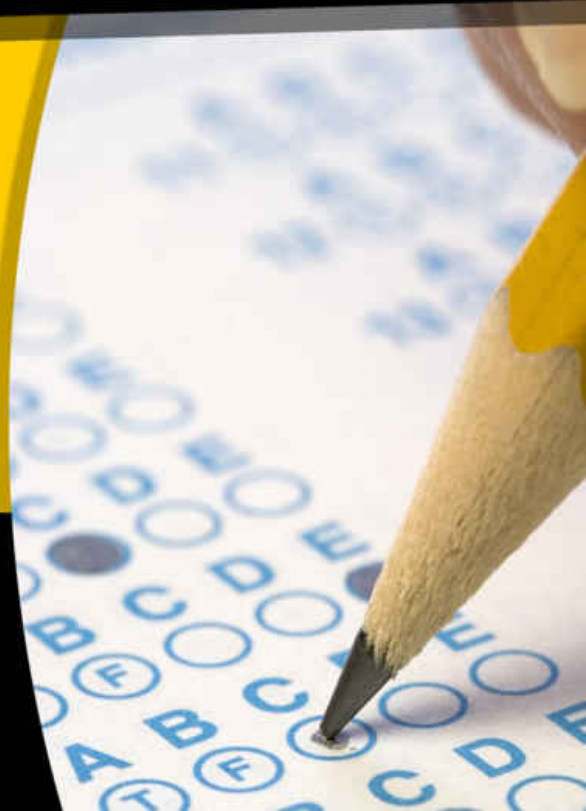
NAVLE Test Review for the North American Veterinary Licensing Examination



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NAVLE Test Review for the North American Veterinary Licensing Examination

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Dear Future Exam Success Story:

First of all, **THANK YOU** for purchasing Mometrix study materials!

Second, congratulations! You are one of the few determined test-takers who are committed to doing whatever it takes to excel on your exam. **You have come to the right place.** We developed these study materials with one goal in mind: to deliver you the information you need in a format that's concise and easy to use.

In addition to optimizing your guide for the content of the test, we've outlined our recommended steps for breaking down the preparation process into small, attainable goals so you can make sure you stay on track.

We've also analyzed the entire test-taking process, identifying the most common pitfalls and showing how you can overcome them and be ready for any curveball the test throws you.

Standardized testing is one of the biggest obstacles on your road to success, which only increases the importance of doing well in the high-pressure, high-stakes environment of test day. Your results on this test could have a significant impact on your future, and this guide provides the information and practical advice to help you achieve your full potential on test day.

Your success is our success

We would love to hear from you! If you would like to share the story of your exam success or if you have any questions or comments in regard to our products, please contact us at **800-673-8175** or **support@mometrix.com** .

Thanks again for your business and we wish you continued success!

Sincerely,
The Mometrix Test Preparation Team

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Written and edited by the Mometrix Exam Secrets Test Prep Team
Printed in the United States of America

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Introduction

Thank you for purchasing this resource ! You have made the choice to prepare yourself for a test that could have a huge impact on your future, and this guide is designed to help you be fully ready for test day. Obviously, it's important to have a solid understanding of the test material, but you also need to be prepared for the unique environment and stressors of the test, so that you can perform to the best of your abilities.

For this purpose, the first section that appears in this guide is the **Secret Keys** . We've devoted countless hours to meticulously researching what works and what doesn't, and we've boiled down our findings to the five most impactful steps you can take to improve your performance on the test. We start at the beginning with study planning and move through the preparation process, all the way to the testing strategies that will help you get the most out of what you know when you're finally sitting in front of the test.

We recommend that you start preparing for your test as far in advance as possible. However, if you've bought this guide as a last-minute study resource and only have a few days before your test, we recommend that you skip over the first two Secret Keys since they address a long-term study plan.

If you struggle with **test anxiety** , we strongly encourage you to check out our recommendations for how you can overcome it. Test anxiety is a formidable foe, but it can be beaten, and we want to make sure you have the tools you need to defeat it.

Secret Key #1 – Plan Big, Study Small

There's a lot riding on your performance. If you want to ace this test, you're going to need to keep your skills sharp and the material fresh in your mind. You need a plan that lets you review everything you need to know while still fitting in your schedule. We'll break this strategy down into three categories.

Information Organization

Start with the information you already have: the official test outline. From this, you can make a complete list of all the concepts you need to cover before the test. Organize these concepts into groups that can be studied together, and create a list of any related vocabulary you need to learn so you can brush up on any difficult terms. You'll want to keep this vocabulary list handy once you actually start studying since you may need to add to it along the way.

Time Management

Once you have your set of study concepts, decide how to spread them out over the time you have left before the test. Break your study plan into small, clear goals so you have a manageable task for each day and know exactly what you're doing. Then just focus on one small step at a time. When you manage your time this way, you don't need to spend hours at a time studying. Studying a small block of content for a short period each day helps you retain information better and avoid stressing over how much you have left to do. You can relax knowing that you have a plan to cover everything in time. In order for this strategy to be effective though, you have to start studying early and stick to your schedule. Avoid the exhaustion and futility that comes from last-minute cramming!

Study Environment

The environment you study in has a big impact on your learning. Studying in a coffee shop, while probably more enjoyable, is not likely to be as fruitful as studying in a quiet room. It's important to keep distractions to a minimum. You're only planning to study for a short block of time, so make the most of it. Don't pause to check your phone or get up to find a snack. It's also important to **avoid multitasking** . Research has consistently shown that multitasking will make your studying dramatically less effective. Your study area should also be comfortable and well-lit so you don't have the distraction of straining your eyes or sitting on an uncomfortable chair.

The time of day you study is also important. You want to be rested and alert. Don't wait until just before bedtime. Study when you'll be most likely to comprehend and remember. Even better, if you know what time of day your test will be, set that time aside for study. That way your brain will be used to working on that subject at that specific time and you'll have a better chance of recalling information.

Finally, it can be helpful to team up with others who are studying for the same test. Your actual studying should be done in as isolated an environment as possible, but the work of organizing the information and setting up the study plan can be divided up. In between study sessions, you can discuss with your teammates the concepts that you're all studying and quiz each other on the details. Just be sure that your teammates are as serious about the test as you are. If you find that your study time is being replaced with social time, you might need to find a new team.

Secret Key #2 – Make Your Studying Count

You're devoting a lot of time and effort to preparing for this test, so you want to be absolutely certain it will pay off. This means doing more than just reading the content and hoping you can remember it on test day. It's important to make every minute of study count. There are two main areas you can focus on to make your studying count:

Retention

It doesn't matter how much time you study if you can't remember the material. You need to make sure you are retaining the concepts. To check your retention of the information you're learning, try recalling it at later times with minimal prompting. Try carrying around flashcards and glance at one or two from time to time or ask a friend who's also studying for the test to quiz you.

To enhance your retention, look for ways to put the information into practice so that you can apply it rather than simply recalling it. If you're using the information in practical ways, it will be much easier to remember. Similarly, it helps to solidify a concept in your mind if you're not only reading it to yourself but also explaining it to someone else. Ask a friend to let you teach them about a concept you're a little shaky on (or speak aloud to an imaginary audience if necessary). As you try to summarize, define, give examples, and answer your friend's questions, you'll understand the concepts better and they will stay with you longer. Finally, step back for a big picture view and ask yourself how each piece of information fits with the whole subject. When you link the different concepts together and see them working together as a whole, it's easier to remember the individual components.

Finally, practice showing your work on any multi-step problems, even if you're just studying. Writing out each step you take to solve a problem will help solidify the process in your mind, and you'll be more likely to remember it during the test.

Modality

Modality simply refers to the means or method by which you study. Choosing a study modality that fits your own individual learning style is crucial. No two people learn best in exactly the same way, so it's important to know your strengths and use them to your advantage.

For example, if you learn best by visualization, focus on visualizing a concept in your mind and draw an image or a diagram. Try color-coding your notes,

illustrating them, or creating symbols that will trigger your mind to recall a learned concept. If you learn best by hearing or discussing information, find a study partner who learns the same way or read aloud to yourself. Think about how to put the information in your own words. Imagine that you are giving a lecture on the topic and record yourself so you can listen to it later.

For any learning style, flashcards can be helpful. Organize the information so you can take advantage of spare moments to review. Underline key words or phrases. Use different colors for different categories. Mnemonic devices (such as creating a short list in which every item starts with the same letter) can also help with retention. Find what works best for you and use it to store the information in your mind most effectively and easily.

Secret Key #3 – Practice the Right Way

Your success on test day depends not only on how many hours you put into preparing, but also on whether you prepared the right way. It's good to check along the way to see if your studying is paying off. One of the most effective ways to do this is by taking practice tests to evaluate your progress. Practice tests are useful because they show exactly where you need to improve. Every time you take a practice test, pay special attention to these three groups of questions:

- The questions you got wrong
- The questions you had to guess on, even if you guessed right
- The questions you found difficult or slow to work through

This will show you exactly what your weak areas are, and where you need to devote more study time. Ask yourself why each of these questions gave you trouble. Was it because you didn't understand the material? Was it because you didn't remember the vocabulary? Do you need more repetitions on this type of question to build speed and confidence? Dig into those questions and figure out how you can strengthen your weak areas as you go back to review the material.

Additionally, many practice tests have a section explaining the answer choices. It can be tempting to read the explanation and think that you now have a good understanding of the concept. However, an explanation likely only covers part of the question's broader context. Even if the explanation makes sense, **go back and investigate** every concept related to the question until you're positive you have a thorough understanding.

As you go along, keep in mind that the practice test is just that: practice. Memorizing these questions and answers will not be very helpful on the actual test because it is unlikely to have any of the same exact questions. If you only know the right answers to the sample questions, you won't be prepared for the real thing. **Study the concepts** until you understand them fully, and then you'll be able to answer any question that shows up on the test.

It's important to wait on the practice tests until you're ready. If you take a test on your first day of study, you may be overwhelmed by the amount of material covered and how much you need to learn. Work up to it gradually.

On test day, you'll need to be prepared for answering questions, managing your time, and using the test-taking strategies you've learned. It's a lot to balance, like a mental marathon that will have a big impact on your future. Like training for a marathon, you'll need to start slowly and work your way up. When test day arrives, you'll be ready.

Start with the strategies you've read in the first two Secret Keys—plan your course and study in the way that works best for you. If you have time, consider using multiple study resources to get different approaches to the same concepts. It can be helpful to see difficult concepts from more than one angle. Then find a good source for practice tests. Many times, the test website will suggest potential study resources or provide sample tests.

Practice Test Strategy

When you're ready to start taking practice tests, follow this strategy:

1. Take the first test with no time constraints and with your notes and study guide handy. Take your time and focus on applying the strategies you've learned.
2. Take the second practice test open-book as well, but set a timer and practice pacing yourself to finish in time.
3. Take any other practice tests as if it were test day. Set a timer and put away your study materials. Sit at a table or desk in a quiet room, imagine yourself at the testing center, and answer questions as quickly and accurately as possible.
4. Keep repeating step 3 on a regular basis until you run out of practice tests or it's time for the actual test. Your mind will be ready for the schedule and stress of test day, and you'll be able to focus on recalling the material you've learned.

Secret Key #4 – Pace Yourself

Once you're fully prepared for the material on the test, your biggest challenge on test day will be managing your time. Just knowing that the clock is ticking can make you panic even if you have plenty of time left. Work on pacing yourself so you can build confidence against the time constraints of the exam. Pacing is a difficult skill to master, especially in a high-pressure environment, so **practice is vital** .

Set time expectations for your pace based on how much time is available. For example, if a section has 60 questions and the time limit is 30 minutes, you know you have to average 30 seconds or less per question in order to answer them all. Although 30 seconds is the hard limit, set 25 seconds per question as your goal, so you reserve extra time to spend on harder questions. When you budget extra time for the harder questions, you no longer have any reason to stress when those questions take longer to answer.

Don’t let this time expectation distract you from working through the test at a calm, steady pace, but keep it in mind so you don’t spend too much time on any one question. Recognize that taking extra time on one question you don’t understand may keep you from answering two that you do understand later in the test. If your time limit for a question is up and you’re still not sure of the answer, mark it and move on, and come back to it later if the time and the test format allow. If the testing format doesn’t allow you to return to earlier questions, just make an educated guess; then put it out of your mind and move on.

On the easier questions, be careful not to rush. It may seem wise to hurry through them so you have more time for the challenging ones, but it’s not worth missing one if you know the concept and just didn’t take the time to read the question fully. Work efficiently but make sure you understand the question and have looked at all of the answer choices, since more than one may seem right at first.

Even if you’re paying attention to the time, you may find yourself a little behind at some point. You should speed up to get back on track, but do so wisely. Don’t panic; just take a few seconds less on each question until you’re caught up. Don’t guess without thinking, but do look through the answer choices and eliminate any you know are wrong. If you can get down to two choices, it is often worthwhile to guess from those. Once you’ve chosen an answer, move on and don’t dwell on any that you skipped or had to hurry through. If a question was

taking too long, chances are it was one of the harder ones, so you weren't as likely to get it right anyway.

On the other hand, if you find yourself getting ahead of schedule, it may be beneficial to slow down a little. The more quickly you work, the more likely you are to make a careless mistake that will affect your score. You've budgeted time for each question, so don't be afraid to spend that time. Practice an efficient but careful pace to get the most out of the time you have.

Secret Key #5 – Have a Plan for Guessing

When you're taking the test, you may find yourself stuck on a question. Some of the answer choices seem better than others, but you don't see the one answer choice that is obviously correct. What do you do?

The scenario described above is very common, yet most test takers have not effectively prepared for it. Developing and practicing a plan for guessing may be one of the single most effective uses of your time as you get ready for the exam.

In developing your plan for guessing, there are three questions to address:

- When should you start the guessing process?
- How should you narrow down the choices?
- Which answer should you choose?

When to Start the Guessing Process

Unless your plan for guessing is to select C every time (which, despite its merits, is not what we recommend), you need to leave yourself enough time to apply your answer elimination strategies. Since you have a limited amount of time for each question, that means that if you're going to give yourself the best shot at guessing correctly, you have to decide quickly whether or not you will guess.

Of course, the best-case scenario is that you don't have to guess at all, so first, see if you can answer the question based on your knowledge of the subject and basic reasoning skills. Focus on the key words in the question and try to jog your memory of related topics. Give yourself a chance to bring the knowledge to mind, but once you realize that you don't have (or you can't access) the knowledge you need to answer the question, it's time to start the guessing process.

It's almost always better to start the guessing process too early than too late. It only takes a few seconds to remember something and answer the question from knowledge. Carefully eliminating wrong answer choices takes longer. Plus, going through the process of eliminating answer choices can actually help jog your memory.

Summary: Start the guessing process as soon as you decide that you can't answer the question based on your knowledge.

How to Narrow Down the Choices

The next chapter in this book (**Test-Taking Strategies**) includes a wide range of strategies for how to approach questions and how to look for answer choices to eliminate. You will definitely want to read those carefully, practice them, and figure out which ones work best for you. Here though, we're going to address a mindset rather than a particular strategy.

Your chances of guessing an answer correctly depend on how many options you are choosing from.

How many choices you have	How likely you are to guess correctly
5	20%
4	25%
3	33%
2	50%
1	100%

You can see from this chart just how valuable it is to be able to eliminate incorrect answers and make an educated guess, but there are two things that many test takers do that cause them to miss out on the benefits of guessing:

- Accidentally eliminating the correct answer
- Selecting an answer based on an impression

We'll look at the first one here, and the second one in the next section.

To avoid accidentally eliminating the correct answer, we recommend a thought exercise called **the $5 challenge** . In this challenge, you only eliminate an answer choice from contention if you are willing to bet $5 on it being wrong. Why $5? Five dollars is a small but not insignificant amount of money. It's an amount you could afford to lose but wouldn't want to throw away. And while losing $5 once might not hurt too much, doing it twenty times will set you back $100. In the same way, each small decision you make—eliminating a choice here, guessing on a question there—won't by itself impact your score very much, but when you put them all together, they can make a big difference. By holding each answer choice elimination decision to a higher standard, you can reduce the risk of accidentally eliminating the correct answer.

The $5 challenge can also be applied in a positive sense: If you are willing to bet $5 that an answer choice *is* correct, go ahead and mark it as correct.

Summary: Only eliminate an answer choice if you are willing to bet $5 that it is wrong.

Which Answer to Choose

You're taking the test. You've run into a hard question and decided you'll have to guess. You've eliminated all the answer choices you're willing to bet $5 on. Now you have to pick an answer. Why do we even need to talk about this? Why can't you just pick whichever one you feel like when the time comes?

The answer to these questions is that if you don't come into the test with a plan, you'll rely on your impression to select an answer choice, and if you do that, you risk falling into a trap. The test writers know that everyone who takes their test will be guessing on some of the questions, so they intentionally write wrong answer choices to seem plausible. You still have to pick an answer though, and if the wrong answer choices are designed to look right, how can you ever be sure that you're not falling for their trap? The best solution we've found to this dilemma is to take the decision out of your hands entirely. Here is the process we recommend:

Once you've eliminated any choices that you are confident (willing to bet $5) are wrong, select the first remaining choice as your answer.

Whether you choose to select the first remaining choice, the second, or the last, the important thing is that you use some preselected standard. Using this approach guarantees that you will not be enticed into selecting an answer choice that looks right, because you are not basing your decision on how the answer choices look.

This is not meant to make you question your knowledge. Instead, it is to help you recognize the difference between your knowledge and your impressions. There's a huge difference between thinking an answer is right because of what you know, and thinking an answer is right because it looks or sounds like it should be right.

Summary: To ensure that your selection is appropriately random, make a predetermined selection from among all answer choices you have not eliminated.

Test-Taking Strategies

This section contains a list of test-taking strategies that you may find helpful as you work through the test. By taking what you know and applying logical thought, you can maximize your chances of answering any question correctly!

It is very important to realize that every question is different and every person is different: no single strategy will work on every question, and no single strategy will work for every person. That's why we've included all of them here, so you can try them out and determine which ones work best for different types of questions and which ones work best for you.

Question Strategies

Read Carefully

Read the question and answer choices carefully. Don't miss the question because you misread the terms. You have plenty of time to read each question thoroughly and make sure you understand what is being asked. Yet a happy medium must be attained, so don't waste too much time. You must read carefully, but efficiently.

Contextual Clues

Look for contextual clues. If the question includes a word you are not familiar with, look at the immediate context for some indication of what the word might mean. Contextual clues can often give you all the information you need to decipher the meaning of an unfamiliar word. Even if you can't determine the meaning, you may be able to narrow down the possibilities enough to make a solid guess at the answer to the question.

Prefixes

If you're having trouble with a word in the question or answer choices, try dissecting it. Take advantage of every clue that the word might include. Prefixes and suffixes can be a huge help. Usually they allow you to determine a basic meaning. Pre- means before, post- means after, pro - is positive, de- is negative. From prefixes and suffixes, you can get an idea of the general meaning of the word and try to put it into context.

Hedge Words

Watch out for critical hedge words, such as *likely* , *may* , *can* , *sometimes* , *often* , *almost* , *mostly* , *usually* , *generally* , *rarely* , and *sometimes* . Question writers insert these hedge phrases to cover every possibility. Often an answer choice

will be wrong simply because it leaves no room for exception. Be on guard for answer choices that have definitive words such as *exactly* and *always* .

Switchback Words

Stay alert for *switchbacks* . These are the words and phrases frequently used to alert you to shifts in thought. The most common switchback words are *but* , *although* , and *however* . Others include *nevertheless* , *on the other hand* , *even though* , *while* , *in spite of* , *despite* , *regardless of* . Switchback words are important to catch because they can change the direction of the question or an answer choice.

Face Value

When in doubt, use common sense. Accept the situation in the problem at face value. Don't read too much into it. These problems will not require you to make wild assumptions. If you have to go beyond creativity and warp time or space in order to have an answer choice fit the question, then you should move on and consider the other answer choices. These are normal problems rooted in reality. The applicable relationship or explanation may not be readily apparent, but it is there for you to figure out. Use your common sense to interpret anything that isn't clear.

Answer Choice Strategies

Answer Selection

The most thorough way to pick an answer choice is to identify and eliminate wrong answers until only one is left, then confirm it is the correct answer. Sometimes an answer choice may immediately seem right, but be careful. The test writers will usually put more than one reasonable answer choice on each question, so take a second to read all of them and make sure that the other choices are not equally obvious. As long as you have time left, it is better to read every answer choice than to pick the first one that looks right without checking the others.

Eliminate Answers

Eliminate answer choices as soon as you realize they are wrong, but make sure you consider all possibilities. If you are eliminating answer choices and realize that the last one you are left with is also wrong, don't panic. Start over and consider each choice again. There may be something you missed the first time that you will realize on the second pass.

Avoid Fact Traps

Don't be distracted by an answer choice that is factually true but doesn't answer the question. You are looking for the choice that answers the question. Stay focused on what the question is asking for so you don't accidentally pick an answer that is true but incorrect. Always go back to the question and make sure the answer choice you've selected actually answers the question and is not merely a true statement.

Extreme Statements

In general, you should avoid answers that put forth extreme actions as standard practice or proclaim controversial ideas as established fact. An answer choice that states the "process should be used in certain situations, if…" is much more likely to be correct than one that states the "process should be discontinued completely." The first is a calm rational statement and doesn't even make a definitive, uncompromising stance, using a hedge word *if* to provide wiggle room, whereas the second choice is a radical idea and far more extreme.

Benchmark

As you read through the answer choices and you come across one that seems to answer the question well, mentally select that answer choice. This is not your final answer, but it's the one that will help you evaluate the other answer choices. The one that you selected is your benchmark or standard for judging each of the other answer choices. Every other answer choice must be compared to your benchmark. That choice is correct until proven otherwise by another answer choice beating it. If you find a better answer, then that one becomes your new benchmark. Once you've decided that no other choice answers the question as well as your benchmark, you have your final answer.

Predict the Answer

Before you even start looking at the answer choices, it is often best to try to predict the answer. When you come up with the answer on your own, it is easier to avoid distractions and traps because you will know exactly what to look for. The right answer choice is unlikely to be word-for-word what you came up with, but it should be a close match. Even if you are confident that you have the right answer, you should still take the time to read each option before moving on.

General Strategies

Tough Questions

If you are stumped on a problem or it appears too hard or too difficult, don't waste time. Move on! Remember though, if you can quickly check for obviously incorrect answer choices, your chances of guessing correctly are greatly

improved. Before you completely give up, at least try to knock out a couple of possible answers. Eliminate what you can and then guess at the remaining answer choices before moving on.

Check Your Work

Since you will probably not know every term listed and the answer to every question, it is important that you get credit for the ones that you do know. Don't miss any questions through careless mistakes. If at all possible, try to take a second to look back over your answer selection and make sure you've selected the correct answer choice and haven't made a costly careless mistake (such as marking an answer choice that you didn't mean to mark). This quick double check should more than pay for itself in caught mistakes for the time it costs.

Pace Yourself

It's easy to be overwhelmed when you're looking at a page full of questions; your mind is confused and full of random thoughts, and the clock is ticking down faster than you would like. Calm down and maintain the pace that you have set for yourself. Especially as you get down to the last few minutes of the test, don't let the small numbers on the clock make you panic. As long as you are on track by monitoring your pace, you are guaranteed to have time for each question.

Don't Rush

It is very easy to make errors when you are in a hurry. Maintaining a fast pace in answering questions is pointless if it makes you miss questions that you would have gotten right otherwise. Test writers like to include distracting information and wrong answers that seem right. Taking a little extra time to avoid careless mistakes can make all the difference in your test score. Find a pace that allows you to be confident in the answers that you select.

Keep Moving

Panicking will not help you pass the test, so do your best to stay calm and keep moving. Taking deep breaths and going through the answer elimination steps you practiced can help to break through a stress barrier and keep your pace.

Final Notes

The combination of a solid foundation of content knowledge and the confidence that comes from practicing your plan for applying that knowledge is the key to maximizing your performance on test day. As your foundation of content knowledge is built up and strengthened, you'll find that the strategies included

in this chapter become more and more effective in helping you quickly sift through the distractions and traps of the test to isolate the correct answer.

Now it's time to move on to the test content chapters of this book, but be sure to keep your goal in mind. As you read, think about how you will be able to apply this information on the test. If you've already seen sample questions for the test and you have an idea of the question format and style, try to come up with questions of your own that you can answer based on what you're reading. This will give you valuable practice applying your knowledge in the same ways you can expect to on test day.

Good luck and good studying!

Activities (Veterinary Practice Roles)

Data Gathering and Interpretation

History, Examination and Environment

Equine Passive Stay Apparatus

The horse has a passive stay apparatus on both the front and rear legs that allows the horse to stand on its feet for a long amount of time without exerting much muscular effort. The stay apparatus consists of tendons and ligaments that stabilize the horse's joints in such a manner that it prevents them from flexing. It consists of three parts: interosseus, proximal sesamoid bones, and sesamoidean ligaments. The shoulder joint, the elbow joint, and the carpal joint are all stabilized or prevented from flexing by pressure exerted on various muscles and ligaments. The pastern and fetlock joints, however, are extended and overextended to prevent the fetlock from drooping to the ground.

Spinal Cord

The spinal cord lies within the spinal canal. The wall of the spinal cord is formed by the vertebra, intervertebral ligaments, and intervertebral discs. The spinal cord is composed of gray matter and white matter. The white matter branches off into roots (ventral and dorsal), which combine to form spinal nerves. The spinal nerves and blood vessels exit the vertebral canal via intervertebral foramina, which are openings between the vertebral arches. The spinal cord itself is covered by three separate membranes. The dura matter lies against the spinal canal. It is composed of two layers: the periosteum (outer layer) and the investing layer. The arachnoid mater is the middle layer and the pia mater is the innermost layer. Between the spinal canal wall and dura mater is the epidural space. Between the arachnoid mater and the pia mater is the subarachnoid space in which the spinal fluid collects.

Types of Bone

Bone is composed of three major substances: organic matrix, osteogenic cells, and minerals. The matrix is composed of collagen and proteoglycans. There are two types of bone matrix: hard cortical bone surrounds marrow in the long bones; cancellous bone is composed of trabeculae (interlacing partitions) that enclose marrow. The flat bones, vertebra, and the ends of the long bones are composed of cancellous bone. The types of cells found in the bone are osteocytes, osteoblasts, and osteoclasts. Osteocytes are found deep in the matrix and function to transport mineral ions and to maintain the bone. Osteoblasts are on the cell surface to control the transport of material in

mineralizing the bone. Osteoclasts are located in the area where the bone is being remodeled and produce acid phosphatase and collagenase to degrade the matrix. The predominant mineral is calcium phosphate.

Bone Healing

The process of bone healing can be loosely described in terms of three stages: an inflammatory stage, a reparative stage, and a remodeling stage. The inflammatory stage occurs immediately after trauma to the bone (usually a fracture). At the fracture site, a hematoma forms and coagulates into a clot. At the fracture ends, the bone is dead. The necrotic tissue causes an inflammatory response; vasodilatation and plasma exudation, which cause edema. This attracts inflammatory cells, leukocytes, and macrophages. The reparative stage starts with hematoma organization in which cells involved in repairing the damage as well as granulation tissue come to the fracture site. Capillary buds (blood vessels) are also observed at this site. A callus is formed, which is eventually replaced by bone. The reparative stage is followed by a long remodeling phase, in which the bone will eventually return to full function.

Position Terms

The following is a partial listing of terms indicating position:

Term	**Meaning**
Abaxial	Lying away from the axis of the body.
Abduction	The withdrawal of a part from the axis of the body.
Adduction	The act of drawing toward the axis of the body.
Anteversion	Turning cranial or inclining cranial.
Axial	Relating to the central part of the body.
Dorsiflexion	Flexion or bending upward of the paw or toes.
Eversion	Turning outward or inside out.
Extension	A movement that brings the members of the limb toward 180°.
External rotation	Eversion.
Flexion	The act of bending from a large angle to a lesser angle.
Hyperextension	Extreme or excessive extension that is greater than 180°.
Internal rotation	Inversion.
Inversion	A turning inward, inside out, upside down, or other reversal of the normal relation of a part.
Oblique	Slanting away from horizontal or perpendicular.
Palmar	Referring to the walking surface of the forepaw.
Pes	Of or referring to the foot or paw.
Plantar	Referring to the walking surface of the hind paw.
Pronation	The act of turning the forepaw outward from the body.
Rotation	The process of turning around an axis.

Valgus	Bent outward away from the midline of the body distal to the joint described.
Varus	Bent inward toward the midline of the body distal to the joint described.
Volar	Referring to the walking surface of the paw.

Mammalian Heart

The heart in a mammal is a four-chambered muscular organ enclosed in a pericardial sac. The pericardial sac is lined with serous membranes; a parietal layer and a serous membrane. The heart wall is made up of three layers: the inner layer, or the endocardium, the middle layer, or the myocardium, and the outer layer, or the epicardium. The four chambers of the heart are called the right atrium, the right ventricle, the left atrium, and the left ventricle. The thin-walled atria receive blood from the veins. The thick-walled ventricles pump blood from the heart: the right ventricle to the lungs and the left ventricle to the body. The four valves of the heart are the atrioventricular valves (the right valve is called the tricuspid valve, the left is called the bicuspid or mitral valve) and the semilunar valves (pulmonary and aortic.)

Flow of Blood

The heart functions to receive deoxygenated blood from the body and to pump oxygenated blood into the body. Deoxygenated blood from the body is pumped through the right atrium to the right ventricle and then to the lungs. After the blood is oxygenated in the lungs, it is pumped through the left atrium to the left ventricle and then to the body. The blood enters the right atrium from the superior and inferior vena cava; it then leaves the right ventricle to the lungs via the pulmonary artery. Pulmonary veins return the oxygenated blood to the left atrium; it is then sent out from the left ventricle via the ascending aorta.

Autonomic Nervous System

The autonomic nervous system is for the most part not under voluntary control. It is an efferent system (except in the case of skeletal muscle) that transmits nervous impulses from the central nervous system to the peripheral organs. Some of its functions are to control heart rate and force, constrict and dilate blood vessels, contract and relax the smooth muscles, perform visual accommodation and control pupil size, and control exocrine and endocrine gland secretion. The autonomic nervous system is divided into two parts: The parasympathetic nervous system reduces blood pressure and heart rate and helps the body digest and absorb nutrients and excrete wastes, whereas the sympathetic nervous system is involved in flight or fight activities, including increasing the heart rate, cardiac output, and blood pressure, diverting blood

flow to skeletal muscles, increasing pupil size, and mobilizing fat and glycogen stores, as well as bronchial dilation and sphincter contraction.

Review Video: Autonomic Nervous System
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The Brain

The brain includes the cerebrum, cerebellum, and the brain stem. The brain stem includes the diencephalon (hypothalamus, thalamus), the midbrain (mesencephalon), the pons (ventral metencephalon), and the medulla oblongata (myelencephalon). Upper motor neurons originate in the brain; they are divided into the pyramidal (cerebral cortex) and extrapyramidal (brain-stem) neurons and control the body's motor activity. The upper neurons stimulate or inhibit the lower motor neurons, which directly innervate the muscles. The function of the upper motor neurons includes regulating the animal's posture, maintaining muscle tone, and initiating the animal's voluntary movements. In dogs and cats, the extrapyramidal system is the dominant upper motor system, while in the primate; the pyramidal system is the dominant upper motor system.

Components of Blood

Blood is composed of plasma and cellular components. The normal percentage of cells is approximately 30 to 45 percent. Plasma is composed primarily of water (90%) and protein (5 to 8 percent); the remainder is made up of gases, hormones, nutrients, electrolytes, and waste products. There are three protein types in the blood: albumin maintains the water in the blood by drawing water into the bloodstream by osmosis, while globulins or antibodies are used as immune system components and to transport other molecules; fibrinogen is needed to help the blood clot. When fibrinogen is removed from the plasma, the resulting product is called serum. The other components of the blood are the platelets, red blood cells, and white blood cells.

Red and White Blood Cells

The red blood cells carry oxygen throughout the body; they are produced in the bone marrow in a process called erythropoiesis. In the dog, the average life span of a red blood cell is 100 to 110 days. In the cat, the life span is 66 to 78 days. Hemoglobin is found in the red blood cell; this iron-containing molecule carries most (97 %) of the oxygen in the body. There are five types of white blood cells (leukocytes) in the blood: neutrophils, lymphocytes, monocytes, eosinophils, and basophils. Neutrophils destroy and clean up pathogens, lymphocytes produce antibiotics used in the immune system, and monocytes

(macrophages in the tissues) use phagocytosis to engulf microorganisms. Eosinophils play a role in allergic reactions and in fighting parasites. Basophils also play a part in allergic reactions and contain histamine.

Renal System

The primary function of the renal system is to excrete nitrogen-containing wastes from the body. When proteins and amino acids are metabolized, ammonia is produced. Because it is toxic to the body, the liver converts ammonia to urea. Another nitrogen waste, uric acid, is produced by the breakdown of nucleotides. Because Dalmatians are not able to break down uric acid efficiently, they may have bladder stones that form from this chemical. Another waste product excreted by the renal system is creatinine, which is produced by the breakdown of muscles. Other functions of the renal system are to regulate sodium, potassium, and chloride levels, maintain the body's water balance and the blood pH, and to control blood pressure and production of hormones for red blood cell formation.

Nephron Filters

The kidneys produce urine by filtering blood in the nephron, which is in close proximity to blood vessels that carry blood to the capillaries in the glomerulus via the afferent arteriole. The pressure within the capillaries then forces water and small particles into the Bowman's capsule. This filtered liquid then goes to the proximal tubule, which reabsorbs any necessary substances such as glucose, vitamins, or amino acids that were filtered out of the capillaries and return them to capillaries around the proximal tubule. Water also returns to the bloodstream by osmosis. The remaining fluid passes through the loop of Henle and the distal tubule, where sodium ions are returned to the body. In the loop of Henle, water is not resorbed; however in the distal tubules, water resorption is regulated according to how much water the body needs to conserve or excrete.

Reproductive and Urinary Tracts

The reproductive tracts (male and female) and the kidneys and ureters are derived from the intermediate mesoderm; the bladder and the urethra develop from the urogenital sinus. The gonads develop in the initial stage from the mesoderm of the urogenital ridge, the coelomic epithelium, and primordial germ cells. The testis-determining factor on the Y-chromosome is what makes the testis develop. Without the Y chromosome, the gonads develop into ovaries. The genital ducts form on the lateral wall of the urogenital ridge and are called paramesonephric ducts. These two ducts join to form the uterovaginal canal, which grows toward the urogenital sinus and protrudes into the urogenital sinus, where it is then called the Müllerian tubercle. The testes produce anti-

müllerian hormone, which causes the degeneration of the paramesonephric ducts.

Reproductive Cycle of Sheep

Sheep have 54 chromosomes and breed throughout the year, although there is some dependence on photoperiods. Most sheep breed in decreasing or short day lengths; domestic sheep have breeding seasons from five to seven months. The estrus cycle averages about 16 days. Estrus is influenced by the presence of the ram and lasts for about 30 hours. After the egg is fertilized, it begins dividing and after about 17 days, attaches to the endometrium. The placenta is diffuse for about 30 days after attachment; however, it becomes a polycotyledonary placenta with cotyledons on the placenta and caruncles on the uterus, forming the placetomes. The average length of gestation is 148 days.

Embryo Transplantation and Placental Development

The embryo attaches to the uterus in domesticated animals at varying days after conception, ranging from 12 days after conception in the cow to 25 to 30 days in the horse. The chorionic vesicle contains the embryo, which is surrounded by the amnion. The allantois forms from the embryo's hindgut and surrounds the amnion, fusing with it to form the allanto-amnion. The allantois also fuses with the chorion to form the allantochorion; this in turn must undergo vascularization before it is ready to assume placental functions. Before the placenta is formed, the embryo is nourished by diffusion of uterine milk through the chorion and amnion. Depending on the species, cotyledons will form or villi will form to supply nutrients from the mother to the fetus.

Nervous System

Very early in the embryo's development, a hallow tube composed of ectodermal neural tissue forms at the dorsal midline. This tube consists of three layers: the inner layer called the ventricular zone (ependyma), the intermediate zone, and the marginal zone. The intermediate zone (or mantle layer) eventually becomes gray matter. The marginal zone later becomes white matter after myelinization occurs. From the tube, the neurons and the glial cells develop. The neural tube is divided into four sections that eventually develop into regions of the central nervous system. The prosencephalon develops into the cerebrum, optic vessels, and the hypothalamus. The mesencephalon develops into the midbrain. The rhombencephalon develops into the medulla oblongata, the pons, and the cerebellum; the spinal cord remains the spinal cord.

Review Video: The Nervous System
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Enzymes

Enzymes are made of protein and are biological catalysts that speed up reactions by lowering the activation energy or stabilizing the transition state; this is accomplished by increasing the concentration of the compounds used in the reaction and by bringing these reactants into proper orientation so that a chemical reaction can occur. Enzymes work by following the induced fit mode: Each enzyme has sites that fit the shape, size, or polarity of reactant. When the reactant binds to the enzyme's active site, the enzyme changes shape, bringing the reactants into a new position so that a reaction can occur. Certain enzymes work better in certain pHs. Some enzymes can be destroyed by certain substrates while other substrates can simply inhibit the enzyme through competitive inhibition or noncompetitive inhibition. Temperatures above 104 ° F typically destroy enzymes.

Urea Cycle

The end product of nitrogen metabolism in mammals is urea. The urea cycle (ornithine cycle) converts ammonia into urea in the liver. Ammonia is a product of oxidative deamination reactions; it must be removed from the body because in even small amounts, it is toxic to the body. The urea produced by the urea cycle is then transported to the kidneys where it is removed from the body in the urine. The reaction formation is as given in the equation: 2 Ammonia + carbon dioxide + 3 ATP yields urea + water + 3 ADP. The amount of urea in the body (bloodstream) is measured by the blood urea nitrogen (BUN) levels. An increased BUN level is termed uremia and occurs with chronic or acute kidney failure, urinary tract blockages, or in congestive heart failure.

Cell Structures

The organelles of the cells are organized structures of the cell itself that exist within the cytoplasm, including the cell membrane, centrioles, endoplasmic reticulum, Golgi apparatus, lysosomes, mitochondria, and the nucleus. Centrioles are needed for cell division; centrioles consist of nine groups of three microtubules that form the spindle needed when the cell undergoes mitosis. The endoplasmic reticulum may or may not have ribosomes attached to its network of flat, tubular vesicular structures that lie within the cytoplasm. The ribosomes function to synthesize proteins. Without ribosomes, the endoplasmic reticulum synthesizes lipids and works to synthesize enzyme processes. The Golgi apparatus functions to package material made within the cell for distribution outside the cell and works with the endoplasmic reticulum to secrete enzymes and hormones.

Arteries and Veins

Arteries have thicker walls than veins and usually have a narrower lumen. Arteries constrict and dilate to control blood flow and have a large amount of muscle in their walls in order to withstand forces produced by the heart pumping blood throughout the body. Veins have thinner walls and wider lumens and "store" blood; they depend upon surrounding muscles to push the blood back to the heart. Both vessels are composed of three layers: the tunica intima (innermost), the tunica media (middle), and the tunica adventitia. The tunica intima has an endothelium and subendothelial connective tissue (presence depends on vessel type). The tunica media is composed of smooth muscle, which is thicker in arteries than in veins of similar size. The tunica adventitia consists of longitudinally arranged collagen fibers; it is usually larger in veins than in arteries.

Esophagus

The esophagus is the tube that delivers the food from the mouth to the stomach. The wall of the esophagus is divided into four areas: the mucosa, the submucosa, the muscularia externa, and the adventitia. The mucosa is the innermost layer and is composed of an epithelium, the lamina propria, and the muscularis mucosae. The submucosa is composed of a thick layer of connective tissue that contains collagen and elastic fibers, fat cells and lymphatic vessels, capillaries, and venules. The muscularis externa is composed of two layers of smooth muscles: an inner layer that constricts the lumen and an outer layer that can shorten the tube. The adventitia is the outer layer composed of collagen. The esophagus epithelium is a stratified squamous type which may or may not be keratinized; the extent of keratinization depends upon the amount of roughage the animal eats.

Stomach of a Monogastric Animal

The stomach in a monogastric animal performs a small amount of digestion to prepare food for entry into the small intestine. The gastric glands lining the stomach secrete substances from the chief cells and parietal cells to mix with the food to form chime; the fundus of the stomach has the most cells. Dogs do not have a nonglandular portion of the stomach. The chief cells produce the proenzyme pepsinogen. In the acidic stomach environment, pepsinogen is converted to the proteolytic enzyme pepsin, which breaks peptide bonds. The parietal cells produce massive quantities of hydrochloric acid. An endocrine cell, the enterochromaffin cell, is also found in the gastric glands; it secretes the hormone gastrin, which promotes pepsinogen production by the chief cells, facilitates stomach wall movements, and encourages secretion of hydrochloric acid by the parietal cells.

Skin

The skin is composed of two divisions: the epidermis and the dermis. The epidermis is made up of keratinized stratified squamous epithelium, which in turn is composed of four regions. The outer surface is the stratum corneum, which is composed of flat plates of keratin called squames; squames slough off and are replaced by cells originating from the deeper layers. The keratin from which the squames are composed is a waterproof, hardy protein formed from cells making up the stratum granulosum, which lies right beneath the stratum corneum. Underneath the stratum granulosum lies the stratum spinosum (spiny layer); this layer is composed of large polygonal cells that attach to one another at cell junctions termed desmosomes. The deepest layer, known as the stratum germinativum, lies below the stratum spinosum. The cells in this layer undergo rapid mitosis to supply the cells that are lost at the skin's surface.

Function of Organelles

The organelles of the cells consist of the cell membrane, centrioles, endoplasmic reticulum, the Golgi apparatus, lysosomes, mitochondria, and the nucleus. Lysosomes are produced by the Golgi apparatus and contain digestive enzymes; they float around the cytoplasm and digest food particles, bacteria, and broken down cellular structures. Mitochondria are the primary sources of the cell's energy. The mitochondria within a cell vary in number depending upon the cell's energy demands. Mitochondria are composed of an outer membrane and an inner membrane. The inner membrane consists of folds to which oxidative phosphorylation enzymes attach. Within the inner cavity, enzymes and coenzymes take energy from nutrients. The nucleus is the main control center of the entire cell and works by controlling cell replication and the chemical reactions that occur; it is made up of the nuclear membrane, nucleoli (one or more), and chromatin.

Cellular Basis for Endotoxins

Extravascular and intravascular mononuclear phagocytes are exposed to the endotoxin. The mononuclear phagocytes release thromboxane, which in turn causes platelets to aggregate and also causes vasoconstriction. Macrophages then release tumor necrosis factor and interleukin-1, which in turn stimulate the body to activate neutrophils and endothelial cells. These cells express high-affinity adhesion molecules, resulting in the margination of the neutrophils that migrate between endothelial cells, and causing vascular and tissue damage through the secretion of toxic oxygen metabolites and proteases. Endothelial cells begin to die. The platelet activation, direct endothelial damage, and cytokine-induced procoagulant activity cause thrombosis of the endothelial surface. The macrophages then churn out nitric oxide and the endothelial cells release prostacyclin. These molecules reduce platelet and neutrophil

aggregation and cause vasodilatation and systemic hypotension, signs of endotoxemia.

Specific Anatomy and Problem Lists

Equine

The Guttural Pouch

The guttural pouch is a unique air-filled diverticula of the Eustachian tubes found only in the horse. The guttural pouch communicates with the middle ear and the pharynx and is divided into medial and lateral compartments by the stylohyoid bone. The guttural pouches are located dorsocaudal to the pharynx, rostrodorsal to the retropharyngeal lymph nodes, and ventral to the atlas. Many important nerves and arteries lie in the lateral walls of the guttural pouches, such as the internal carotid artery, branches of the external carotid artery, the cranial sympathetic nerve trunk, the facial nerve (CN VII), the glossopharyngeal nerve (CN IX), the vagus nerve (CN X), the spinal accessory nerve (CN XI), and the hypoglossal nerve (CN XII).

Dental Examination

The examination of the horse's oral cavity should be performed in a methodical manner, using caution to properly restrain the horse and using sedation if necessary and to protect bystanders:

- The mouth should be rinsed with clean water. When rinsing, inspect the nature of the material flushed from the mouth.
- Observe the tongue for wounds, sores, or scars
- Visually examine the incisors for wear. Note any missing or malformed teeth.
- The interdental space should be visually inspected and palpated for canine teeth, unerupted canine teeth, and for wolf teeth (rarely found in the lower arcade)
- Examine the mandible and palpate the oral cavity
- A full-mouth speculum and a light source should be used to visually inspect the inner aspects of the teeth, paying careful attention to gingival pockets or uneven wear or damage to the cheek teeth

Equine Laminitis

Laminitis, while appearing as a hoof and foot problem, is really a sign of a general metabolic condition. The most frequent cause of laminitis is a high carbohydrate diet either through the feeding of too much grain or from eating lush grass or legumes. Another common cause is working poorly conditioned animals too hard. The resulting hard forces on the feet can incite a case of

laminitis. Laminitis can also be a secondary manifestation of other infectious diseases such as colic, enteritis, endotoxemia, or a reaction to overdosing of certain medications, especially corticosteroids; this may lead to a brief ischemia, resulting in the degeneration and failure of the union between the sensitive and horny laminae of the hoof. In more serious cases, the pedal bone may even perforate the sole.

Exertional Myopathy

Exertional myopathy, also called tying up or rhabdomyolysis, is caused by a necrosis of the striated skeletal muscle, usually seen after the horse has started to exercise, and can be found in all breeds of horses. While there are different degrees of clinical signs, many horse afflicted with exertional myopathy show excessive sweating in relation to the amount of work being done, refusal to continue to exercise, muscle pain, cramping, or fasciculations, tachypnea, tachycardia, and painful lumbar and gluteal muscles. Depending on degree of muscle damage, which is shown by increased levels of lactate dehydrogenase, AST, or serum CK, the horse may become recumbent and the urine may become discolored. The disease can either be sporadic or chronic. The chronic form tends to be a genetic disease of specific breeds of horse.

Intraluminal Esophageal Obstruction Ramifications

Intraluminal esophageal obstruction, also known as esophageal feed impaction or choke, frequently occurs in the horse. Greedy eating, poor teeth, low-quality feed, and lack of water (especially in cold weather) all contribute to this problem. Most frequently, food becomes impacted just cranial to the thoracic inlet and a bolus can be palpated. Once the obstruction has been relieved through sedation to relax the esophagus and occasionally lavage via a nasogastric tube, the horse is usually placed on broad-spectrum antibiotics and anti-inflammatory agents. Ulceration of the esophagus can lead to strictures at the site of the obstruction, causing narrowing of the esophagus. There is an increased risk for reoccurrence in the month following an obstruction. Soft feeds or grass should be fed and the horse should be monitored while eating during this time span.

Potomac Horse Fever

Potomac Horse Fever is caused by *Neorickettsia risticii* (formerly known as *Ehrlichia risticii*), which infects the small and large intestinal enterocytes. This disease is more prevalent in the warmer seasons and has been correlated with horses on pasture being infected with *Neorickettsia risticii* that borders streams.

Signs of Potomac Horse Fever include the following:

- Mild depression and anorexia

- Fever between 102-107 °F (38.9-41.7°C)
- Decrease in gut sounds, followed in 24-36 hours by diarrhea, sometimes very severe or watery, along with some signs of colic. Diarrhea can lead to dehydration.
- A few horses develop laminitis or a serious toxemia
- CBC findings indicate neutropenia and lymphopenia, not necessarily correlating with severity of clinical signs
- Pregnant mares may abort after a bout of Potomac Horse Fever

Adult Horse Diarrhea

Causes of adult horse diarrhea range from bacterial infections, viral infections, parasitic infestations, or chemical or toxin-induced diarrhea. When performing a work-up for a horse with diarrhea, the following disease conditions should be kept in mind: Salmonellosis, clostridial overgrowth (*difficile* or *perfringens A*), Cyanthostomiasis, medications (especially overuse of antibiotics), sand ingestion, overfeeding or sudden change in diet, Giardiasis, Potomac Horse Fever, *Mycobacterium avium* infection, enteric lymphosarcoma, disrupted colonic flora, blister beetle ingestion, peritonitis, or very rarely, *Lawsonia intracellularis* (most frequently in yearlings and younger horses), and Equine viral arteritis infection.

Some causes, especially Mycobacterium avium and lymphosarcoma, result in a more chronic course of diarrhea, while others, such as Salmonellosis, clostridia, Potomac Horse Fever, and blister beetle ingestion, may result in a more sudden onset.

Hepatic Disease

Hepatic disease occurs frequently in horses, which can cause hepatic failure, dysfunction, or obstructive biliary disease. Causes of **obstructive biliary disease** in the horse include: Pancreatic disease, inflammation or neoplasia can obstruct the ducts, duodenal ulcerations can cause inflammation where the ducts drain into the digestive tract, intestinal displacement causing functional blockage or inflammation, Aflatoxicosis, liver abscesses or neoplasia, leukoencephalomalacia, alsike clover or kleingrass poisoning, and portal caval shunts or perinatal herpesvirus 1.

Liver failure can be caused by the following conditions or diseases:

Tyzzer's disease, Theiler's disease, Chronic hepatitis, Various toxicosis (pyrrolizidine alkaloid, fumarate, corticosteroids, inhalant anesthesia), Suppurative cholangitis, Cholelithiasis, Hepatic lipidosis, Parasite infestation, Endotoxemia, and endo.

Viral and Bacterial Pneumonia

Viruses are responsible for many cases of pneumonia or respiratory disease in the horse; they also set the stage for a secondary infection by bacteria. Some common viruses which have a predilection for the respiratory tract include equine influenza, equine herpesvirus type 4 (EHV-4), and equine viral arteritis. Other viruses causing less severe lung infection are reovirus, equine rhinitis virus, adenovirus (usually in immune-deficient Arabian foals), and equine herpesvirus type 2. Hendra virus, a zoonotic virus, has caused fatal disease in Australian horses. Bacterial infections usually occur after a viral infection; however, the disease does not follow this pattern. *Streptococcus equi zooepidemicus* is the most common culprit. Other opportunists include *Bordetella bronchiseptica* , *E. coli* , *Pasteurella* , *Actinobacillus equuli* , and *Pseudomonas* . Foals less than six months of age contract pneumonia from *Rhodococcus equi* .

Rhodococcus Equi Infection in Foals

Young foals (one to five months of age) are afflicted by this severe pneumonia. *Rhodococcus equi* is a gram-positive, facultative intracellular bacteria found in the soil. Certain factors favor the spread of the pathogen: high temperatures, dust, and sandy soil. Foals become infected after inhaling the bacteria, which multiplies in the intestine. The stools from foals with replicating *Rhodococcus equi* contaminate the environment. The bacterium grows slowly, and when clinical signs appear, they can be dramatic. Foals appear sick, with a fever and lack of energy. Diarrhea due to intestinal abscesses is common, and upon chest auscultation, the examiner will hear wheezing sounds and crackles. Therapy should commence using erythromycin and rifampin or using azithromycin and rifampin, typically for two months. Other treatment (NSAIDs, IV therapy, lung therapy) should be used as needed.

Causes of Foal Diarrhea

Diarrhea in foals falls into three broad categories: foal heat, viral-induced, and those caused by bacteria. Foal heat occurs when the dam undergoes her first estrus cycle postpartum, usually four to 14 days after the foal's birth. The cause is unknown and it does not debilitate the foal. The most common virus causing foal diarrhea is the rotavirus, which destroys the cells on the tip of the small intestinal villi, leading to malabsorption of feed. Younger foals have the most severe signs, and treatment is supportive until the intestine can regenerate villi tips. The common bacterial pathogens involved in foal diarrhea are *E. coli* , *Salmonella* , Clostridial species, and *Klebsiella* . Appropriate antibiotics should be used following culture and sensitivity testing in order to combat these pathogens, along with appropriate supportive care.

Congenital Heart Defects

Horses are commonly afflicted by four types of congenital heart defects; these include tetralogy of Fallot, tricuspid atresia, patent ductus arteriosus, and ventricular septal defect. The Arabian breed has a higher risk of having a foal with a congenital heart defect than the other horse breeds.

There are many factors that contribute to these heart defects. Genetics or heredity plays a role in some defects. In utero assaults such as infections, toxins, medication given to the mare, poor nutrition during pregnancy, and other environmental factors can all cause a foal's heart to be damaged during crucial periods of development. Young foals may have a heart murmur, which usually disappears by the time the animal is six months old. These innocent murmurs should be disregarded unless other signs of cardiovascular disease are present.

Duodenitis-Proximal Jejunitis

Duodenitis-proximal jejunitis (DPJ) is a disease causing inflammation and edema of the duodenum and proximal jejunum. It is considered to be an idiopathic and sporadic condition and produces these signs: depression, endotoxemia, colic, and ileus. Fluid tends to accumulate in the stomach and small intestine, and other problems can occur such as laminitis, liver damage, and heart arrhythmias. The cause is unknown, although mycotoxins, clostridia, and *Salmonella* have all been investigated as causative agents. The disease can take either an acute or chronic course. In acute cases, supportive therapy is needed, such as placing a nasogastric tube to decompress the stomach and drain accumulated fluid, IV therapy to replace fluids and electrolytes, pain control, and antibiotics. Chronic cases may require surgical intervention by performing an intestinal bypass by either a duodenojejunostomy or a duodenocecostomy.

Cholelithiasis

Cholelithiasis most commonly afflicts horse from six to 15 years of age. Neither sex nor any particular breed is more susceptible. The calculi can cause biliary obstruction in the common bile duct or intrahepatic bile ducts. The cause is unknown, although an ascending biliary tract infection, parasites in the common bile duct, or a change in bile composition have been postulated to cause stone formation. Clinical signs may be intermittent and can vary. Signs can be nonspecific and include abdominal pain, weight loss, icterus, depression, or fever. Occasionally, hepatic failure may be seen. Laboratory results can include increased serum GGT activity, hyperbilirubinemia, leukocytosis, increased serum total bile acid concentration, anemia of chronic disease, hyperproteinemia, hyperfibrinogenemia, hyperglobulinemia, decreased potassium levels, decreased serum urea nitrogen, and decreased glucose.

Guttural Pouch Mycosis

Guttural Pouch Mycosis is most commonly caused by the fungal organism *Aspergillus* spp. The fungus form mycotic plaques in the medial guttural pouch directly over the internal carotid artery. The plaques themselves do not cause problems, but only when they damage the arteries and cranial nerves in the mucosa that lines the pouch. The fungus erodes the wall of the internal carotid artery or branches of the external carotid artery. This causes epistaxis, the most common sign associated with guttural pouch mycosis. Bleeding can be severe and on occasion leads to fatal bleeding. Bleeding can be stopped by occluding the affected artery by a balloon-tipped catheter. If the fungus damages the cranial nerves, Horner's syndrome or dysphagia may develop (this is a poor indicator for success of therapy). The damage may also displace the soft palate dorsally. Treatment is based on sensitivity testing, and is aimed at destroying the fungus through topical and systemic antifungals.

Neonatal Septicemia

Foals suffering from septicemia and associated septic shock become sick due to bloodborne bacteria and their toxins. The primary pathogens involved in this systemic disease are the gram-negative bacteria such as *Pseudomonas* , *Enterobacter* , *E. coli* , *Actinobacillus* , and *Klebsiella* . These bacteria, sometimes along with a concurrent gram-positive bacterial infection, gain access to the blood through infections involving the digestive tract, respiratory system, the navel, or even the placenta. The signs and symptoms shown by a foal with septicemia are attributed to the release of endotoxins. These signs include vasodilatation, fever, bradycardia, hypoglycemia, blood clotting problems, and perhaps disseminated intravascular coagulation. The main risk factor for a foal to contract septicemia is either lack of an adequate amount of colostrum or the dam producing low-quality colostrum. An unsanitary environment, poor mare health, and premature birth compound the lack of quality colostrum.

Verminous Arteritis

Verminous arteritis is a syndrome resulting in ischemic infarcts of the bowel because of thrombi blocking the blood vessels that serve the intestinal tract. The inciting cause of verminous arteritis is an infection of the nematode *Strongylus vulgaris* . This parasite is ingested by the horse as an L3 larva, which reaches the large intestine to burrow into the submucosa. L3 molts into L4 larvae, which then migrates along the intestinal arteries to eventually reach the root of the cranial mesenteric artery. Here, they molt into immature adults and cause a severe arteritis. This damage to the arteries causes the architecture of the artery to change, producing new tracts and aneurysms, and causes thrombi to form, leading to infarcts of parts of the intestine and associated pain and

toxemia. The immature adults return via the bloodstream to the large intestine to complete their life cycle.

Equine Granulomatous Enterocolitis

Equine granulomatous enterocolitis, also known as chronic inflammatory bowel disease, is an infiltrative disease with no definitive cause. The involved portions of the intestinal tract cause loss of fluid and protein (small intestine) or a loss of fluid and electrolytes (large intestine), leading to a malabsorption of nutrients. The diagnosis is extremely difficult and relies upon physical examination, biopsy, clinical signs, and laboratory testing. Treatment is usually unsuccessful and many animals are euthanized, with definitive diagnosis being made at necropsy. Clinical signs include cachexia, along with related skin (hair loss, rough coat, flaky skin) and cardiac signs (murmurs), anorexia, chronic diarrhea, bouts of colic, depression, lymphadenopathy, edema, and nasal/eye discharge. Laboratory testing findings include anemia, hypoproteinemia, hypoalbuminemia, neutrophilia, and abnormal glucose tolerance testing.

Colic

Colic in horses can be caused by a number of conditions. Gastric dilatation is a frequent cause of colic and is caused by excessive gas formation or by an intestinal obstruction. If gastric dilatation is not treated, it can lead to gastric rupture. The stomach may become impacted with feed (gastric impaction) due to poor quality feed, poor teeth condition, and lack of water; however, gastric impaction is an infrequent cause of colic. The small intestine can produce signs of colic due to inflammation, obstruction, or strangulation. Some small intestinal conditions that lead to colic are intussusceptions, proximal enteritis-jejunitis, ileal impaction, adhesions, ascarid impactions, intestinal incarceration, pedunculated lipomas, volvulus, and inguinal hernias. The cecum and large intestine may also cause colic. Impaction of colon contents and foreign bodies, volvulus, enterolithiasis, and left and right dorsal colon displacements may all affect the horse's large intestine.

The mucous membranes (gums, vulva) need to be assessed for color, capillary refill time, and moistness (dehydration). The abdomen should be auscultated in all four quadrants. The left side corresponds to colon (lower) and small intestine (upper). The cecum is on the right. Respiratory rate should be measured. The horse's temperature should also be measured and legs/feet checked for signs of laminitis. A nasogastric tube should also be passed to see if fluid refluxes, as horses do not vomit. The fluid color, composition, and volume should be noted if any fluid refluxes from the stomach. The final, but very important, step is the rectal examination. These structures should be identified and palpated: cecal base and ventral cecal band, duodenum, aorta, cranial

mesenteric artery, peritoneal surface, pelvic flexure, bladder, spleen, left kidney, inguinal rings (males), and ovaries and uterus (mares).

The physical examination of a horse suffering from colic can lend important clues to the cause of the condition. There are four primary reasons a horse develops colic: the intestinal tract becomes inflamed, the intestinal wall is stretched by ingesta, gas, or fluid, a part of the intestine twists or become incarcerated, or increased tension has been placed upon the mesentery. The mucous membrane can be pale in color or hyperemic depending on cardiovascular status. The capillary refill time usually becomes prolonged. A dehydrated horse will have dry mucous membranes. Pain causes the heart and respiratory rates to increase. Nasogastric reflux can be caused by small intestinal obstruction or lack of peristalsis due to ileus. Ileus can also cause a lack of gut sounds, as can ischemia. Other intestinal sounds can indicate an obstruction or distention.

Review Video: Colic
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COPD

COPD, otherwise known as heaves, is an allergic respiratory disease of horses that causes the obstruction of the small airways in the lung and bronchoconstriction. This restriction along with mucus production causes the clinical signs of dyspnea, coughing, and nasal discharge and also limits a horse's ability to exercise. Lung auscultation reveals crackles, wheezes, tracheal rattle, and a prolonged expiration. In less severe cases, the horse may appear normal at rest, but exercise will induce the signs associated with COPD. COPD is especially prevalent in horses that are stabled with straw bedding and fed hay. Removing horses from these allergens greatly decreases the signs and symptoms of COPD. Because the horse has difficulty with expiration, the abdominal muscles can become hypertrophic and develop what is termed a heave line. Treatment consists of limiting exposure to allergens and administration of bronchodilating medication and corticosteroids.

Equine Protozoal Myeloencephalitis

EPM is one of the most commonly diagnosed equine central nervous system (CNS) diseases in the USA. *Sarcocystis neurona* is the usual pathogen that causes EPM, although *Neospora caninum* and *Neospora hughesi* cause EPM symptoms as well. It is usually assumed *Sarcocystis neurona* is the cause of EPM and its host is the opossum. Opossums shed the sporocysts in their feces, which may contaminate feed or water ingested by a horse. The horse is considered to be an

aberrant or dead-end host of *Sarcocystis neurona* ; in the intermediate host, the sporocysts develop into sarcocysts and become embedded in the skeletal muscle. The opossum then consumes infected skeletal muscle, completing the life cycle of *Sarcocystis neurona* .

Tetanus

Second to humans, the horse is the animal most susceptible to tetanus infection. After inoculation, the incubation period is typically 10 to14 days. The first signs of stiffness typically occur around the area of the wound, then after 24 hours, the stiffness spreads to the rest of the body. Spasms, often violent, develop; the horse presents with a prolapsed third eyelid, dilated nostrils, and sweating and its ears are erect. Normal movement is difficult. The tail is stiff and held in an extended position; along with extension of the neck and head and leg stiffness, this gives the appearance of a sawhorse stance. It is difficult for the horse to eat and drink due to spasms of the head muscles. Physical examination reveals increased heart and respiratory rates and congestion of the mucous membranes. The temperature usually does not increase much above normal.

Cervical Vertebral Stenotic Myelopathy

Cervical vertebral stenotic myelopathy, or wobbler syndrome, is caused by a compression of the spinal cord at the cervical level. This compression leads to an abnormal gait in the limbs depending on the level at which the cord is compressed. Clinical signs may include a stiff neck, stumbling, misstepping, appearing wobbly when moving, reluctance to move or rise, falling, or an appearance of being weak. All these signs are actually due to a proprioceptive deficit. Cervical compression can be labeled either dynamic or static; with dynamic compression, the bending of the horse's neck causes stenosis of the intervertebral space between the third and fourth cervical vertebra (older horses) or between the fourth and fifth cervical vertebra (yearlings) and compression of the spine. Static compression is permanent and usually occurs between the fifth and sixth cervical vertebra, or in older horses, the six and seventh cervical vertebra.

Influenza

Equine influenza has a very short incubation period of one to three days and usually occurs in younger horses from one to three years of age. Horses inhale the virus, which attaches to and reproduces in the epithelial cells of the respiratory tract. It is caused by the influenza virus which has a number of subtypes and belongs to the orthomyxovirus group. A horse suffering from equine influenza usually comes down with a sudden high fever that is often biphasic, a cough, and nasal discharge. Along with these signs, a horse may have muscle soreness, pharyngitis, and tracheitis. A horse displaying signs of fever

and nasal discharge may also be infected with equine viral arteritis, strangles, equine herpesvirus (EHV)-1, or EHV-4.

Herpesvirus

Equine herpesvirus 1 (EHV 1) and equine herpesvirus 4 (EHV 4) are the two most important herpesviruses affecting the horse's respiratory system. A horse exposed to the virus inhales the viral particles, which attach to and reproduce in the epithelial cells of the upper respiratory tract (tonsils, nares, and pharynx). After a three- to seven-day incubation period, the virus is then transported by T-lymphocytes to other organs and to the lymph nodes. The virus produces a leucopenia that becomes mucopurulent, along with signs of high fever and a serous nasal discharge. EHV-1 also causes complications with pregnancy (neonatal deaths and abortions), as well as neurological problems. Vaccination is successful in controlling the disease and is typically coupled with the equine influenza vaccine; however, EHV vaccination is typically short-lived and vaccination may need to be boosted in two to three months.

Smoke Inhalation

The damage to the horse's respiratory system is caused by both heat and toxic gases. The gases can cause damage due to the nature of the chemicals from which they are composed or they can combine with water to form acids that exert a corrosive effect on respiratory tract tissues. Along with the toxic gases, carbon monoxide combines with hemoglobin to form carboxyhemoglobin. This leads to hypoxia and decreases tissue oxygen levels. The heat damages the tissues of the upper respiratory tract, which becomes edematous and inflamed. Further damage occurs deeper in the tract, such as obstruction from inflammatory cells, necrosis of cells, edema, and blood pooling. The horse suffers from severe bronchopneumonia and bronchiolitis. In addition, macrophages do not function properly, predisposing the horse to further infections.

Encephalitis

Insects are the primary transmitters of the viruses of the Togaviridae family. These viruses are classified as alphaviruses and cause diseases such as Western, Eastern, and Venezuelan equine encephalomyelitis. In the early stages (three to four days after infection), the clinical signs of these three diseases include anorexia, fever, lethargy, excessive sleepiness, and stiffness. Later, the horse may develop other signs such as ataxia, proprioceptive deficits, aggression, excitability, or other signs contributing to CNS disturbance. Towards the end of the infection, the horse may become comatose and recumbent. If this occurs, death will commence within five days. There is a high mortality rate associated

with infection. Recovered horses usually have residual neurological effects that vary depending on the amount of damage caused by the virus.

Rabies

The rabies virus is in the Rhabdovirus family, genus *Lyssavirus,* and is a fatal neurological disease of warm-blooded animals. The signs attributed to the disease occur anywhere from three weeks to six months after exposure to the rabid animal. If the horse is bitten around the face or neck, the incubation time is shorter. Early in the disease course, the signs may be variable, such as slight ataxia, loss of appetite, or depression. Some horses may act as though they have colic. As the disease progresses to the aggressive form, other signs include behavioral changes, aggression, and pruritis. In the dumb form, the animal appears lethargic, ataxic, depressed, or recumbent. The paralytic form of the disease manifests with ataxia or lameness followed by paraplegia and decreased reflexes and muscle tone.

Fungal Keratitis

A horse can contract fungal keratitis when a damaged cornea becomes seeded with a fungus. The most common culprit involved in fungal keratitis is *Aspergillus.* A typical case presents as a corneal ulcer that is non-healing despite treatment with ophthalmic preparations of antibiotics and steroids. An equine eye with fungal keratitis appears reddened with excessive tearing, causing pain, spasms, and light shyness. The eye may appear hazy and neovascularization of the cornea may occur. Corneal biopsy or scraping assists in making the diagnosis. The aim of treatment is to rid the eye of the fungus by use of antifungal agents formulated for use in the eye. It may be necessary to treat the eye several times a day for several weeks. Cases of deep fungal infection can be surgically removed by a lamellar keratectomy.

Theiler's Disease

Theiler's disease, also known as serum hepatitis, is an acute hepatitis of horses that occurs after administration of equine immune serum. It can occur one to three months after foaling in mares that have been administered tetanus antitoxin. The signs occur suddenly and include icterus, anorexia, photodermatitis, intravascular hemolysis, and central nervous system signs attributed to hepatic encephalopathy (depression, incoordination, behavioral changes, tremors, yawning, and stupor). Serum chemical levels of bilirubin, aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), and sorbitol dehydrogenase (SDH) increase. The GGT levels are particularly prone to early elevations. Liver biopsy can help identify the disease. Treatment is the same as that for animals with hepatic insufficiency.

Tyzzer's Disease

Tyzzer's disease is a disease of foals from one to six weeks of age, often occurring at times of physiologic stress. The responsible organism, *Clostridium piliforme* , causes an acute focal hepatitis. The spores of *C. piliforme* can live in the environment for up to a year. The disease can manifest as a sudden death or present with clinical signs of anorexia, depression, diarrhea, fever, icterus, seizures, and coma. Blood tests reveal increased bilirubin, fibrinogen, and liver enzyme levels (SDH, AST, AP, LDH, and GGT) along with metabolic acidosis. Diagnosis is usually made at necropsy, as almost all foals die once they contract the disease. Treatment in surviving foals is primarily support with fluid therapy and antibiotics.

Acute Renal Failure

Acute renal failure in the horse is caused by toxins or ischemia leading to acute tubular necrosis. One toxin that causes acute tubular necrosis in the horse is aminoglycosides. In order of toxicity to the kidney, these include neomycin, gentamicin, kanamycin, amikacin, and streptomycin. Overdosing is usually not the issue; rather, it is a cumulative effect and/or dehydration due to disease at the time of administration. Vitamin K, heavy metals, acorn ingestion, and nonsteroidal anti-inflammatory drugs (NSAIDs) can also damage the kidneys. High doses of NSAIDs adversely affect the kidney; even with prolonged use, at normal doses they do not generally cause kidney problems. Horses that have suffered an episode of myopathy (such as tying-up syndrome) can also develop acute tubular necrosis.

Pyrrolizidine Alkaloid Toxicity

Pyrrolizidine alkaloid toxicity occurs when horses consume plants containing pyrrolizidine alkaloids. Plants containing this substance include: common groundsel, riddell groundsel, thread leaf groundsel, tansy ragwort, rattlebox, fiddleneck, tar weed, common heliotrope, comfrey, and hound's tongue. The disease is a chronic, progressive liver failure. Horses exhibit signs related to liver failure, including icterus, ataxia, and weight loss. Some horses develop photosensitization of unpigmented areas of the skin. Diagnosis includes taking an accurate nutritional/pasture history and serum chemistry testing, paying particular attention to liver enzymes; these enzymes are elevated in the initial stages, but some may return to normal as the disease progresses. Liver biopsy will greatly assist in making an accurate diagnosis.

Herpesvirus Encephalomyelitis

Herpesvirus encephalomyelitis is caused by the infection of the central nervous system by the equine herpesvirus type 1. A horse that is infected by this organism will display sudden signs due to hemorrhage and edema in the central

nervous system; these include conscious proprioceptive deficits, tetraplegia, fever, tetraparesis, urinary incontinence, perineal desensitization, and a flaccid anus. Males may also develop paraphimosis. The urinary bladder may become distended and cause signs of colic. Ataxia occurs in the hind limbs and the horse may assume a dog-sitting position. Some horses eventually go into a coma and die; other horses have residual effects from the brain swelling, while some make a complete recovery.

Diaphragmatic Hernia

A diaphragmatic hernia happens when there is a break in the diaphragm that allows abdominal organs to enter into the chest cavity. The diaphragm of the horse develops from a fusion of the body wall, the septum transversum, the pleuroperitoneal membranes, and the esophageal mesentery. If any of these structures fail to fuse, the foal may be born with a diaphragmatic hernia. In addition, the forces exerted on the foal's body during birth can also cause rupture of the diaphragm. A diaphragmatic hernia is a rare cause of colic in the mature horse. A ruptured diaphragm may result from a direct blow to the abdomen, very strenuous exercise, severe dystocia, breeding, or severe intestinal distension.

Diaphragmatic hernia occurs when a break in the diaphragm allows intestinal contents to enter the thoracic cavity. The most typical sign of a diaphragmatic hernia in the horse is gastrointestinal distress. Acute severe colic may result from the incarceration and displacement of the intestinal organs into the chest. The horse may also experience respiratory distress caused by the pressure of the abdominal organs in the chest. The intestines may also strangulate when passing thorough the rent in the diaphragm. It may be difficult to diagnose a diaphragmatic hernia in the horse. Intestinal sounds may be heard in the thorax, but these may be referred sounds from the abdomen. Heart sounds may be muffled, but this is not always heard in horses suffering from this condition. Rectal exam may reveal an empty abdomen. The only available treatment option is surgery.

Laryngeal Hemiplegia

Laryngeal hemiplegia (roaring or recurrent laryngeal neuropathy) is caused by a permanent paralysis of the left vocal fold and arytenoid cartilage; it is uncommon for the right side to be affected. Laryngeal hemiplegia is a congenital disorder causing degeneration of the recurrent laryngeal nerves, peroneal nerves, and the long fibers of the CNS. Other causes include perivascular injection of irritating substances, trauma to the recurrent pharyngeal nerve or the vagus nerve, ingestion of chick peas or *Lathyrus* spp. plants, or intoxication from lead or organophosphates. Large-breed horses,

such as draft horses or Thoroughbreds, those with long necks, and males seem predisposed to develop laryngeal hemiplegia. The intrinsic laryngeal muscles atrophy, except for the cricothyroid muscle, which obtains its innervations from the cranial laryngeal nerve; clinical signs are attributed to atrophy of the dorsal cricoarytenoid muscle.

Laryngeal hemiplegia causes inspiratory stridor, characterized by a roaring or whistling sound, usually due to degeneration of the left recurrent laryngeal nerve. This results in loss of control of the abductor muscles in the pharynx, leading to collapse of the arytenoid cartilage and vocal fold. The glottal cross-sectional area is compressed and impedes the flow of air passage. In order to maintain air flow during exercise, the accessory muscles used during breathing need to work harder. This further collapses the left arytenoid cartilage, drawing it close to the unaffected arytenoid cartilage and completely blocking the airway. The roaring or whistling sound is the result of air being forced across the collapsed cartilage and vocal folds.

Horses affected by laryngeal hemiplegia usually do not have noisy breathing when they are resting. When the horse begins to exercise, the characteristic whistling and/or roaring during inspiration occurs; frequently, the horse is not able to tolerate exercise. In addition to these clinical signs, horses with suspected laryngeal hemiplegia should be immediately examined postexercise via endoscopy to monitor the movement of the arytenoid cartilage. Another option is to perform the exam as the horse is placed on a treadmill. Other tests that assist in diagnosis include the laryngeal adductory reflex test (slap test), observation of the arytenoid movement during swallowing and when the nasal passages are occluded, and the arytenoid palpation test. Surgical treatment options include laryngeal ventriculectomy and prosthetic laryngoplasty

Osteochondrosis

Osteochondrosis (OCD) is caused by a defect in endochondral ossification when the foal is developing; this defect occurs when the cartilage matures and produces a cartilage flap, subchondral bone cyst, or physitis. OCD is usually diagnosed when the horse begins to train. The horse exhibits varying degrees of lameness, followed by effusion in the affected joints. The disease is best detected through radiographs; however, lameness and effusion may be present for months before any change is identified. The affected joint may be injected with an anesthetic that masks the pain. Other diseases to consider before making a diagnosis of OCD are trauma, septic arthritis, and synovitis.

Angular Limb Deformity

Foals can be born with angular limb deformities or they can acquire an angular limb deformity after birth. Only one leg can be affected or all four limbs can be deformed. An angular limb deformity is defined as forelimb or hindlimb deviation in the frontal plane axis. A varus deformity is a medial deviation of the limb distal to the deformity's origin; a valgus deformity is defined as a lateral deviation of the limb distal to the deformity's origin. Causes of angular limb deformities include malposition of the fetus in the uterus, poor conformation, trauma, excessive joint laxity, hypothyroidism, or a defect in the endochondral ossification of the long bones, tarsal bones, or the carpal bones.

Tenosynovitis

Tenosynovitis occurs in the horse when the membrane around the tendon sheath becomes inflamed, causing the entire tendon sheath to swell. There are four categories of tenosynovitis: Acute tenosynovitis usually occurs because of trauma and involves a rapid swelling of the tendon sheath along with pain, warmth, and lameness. Chronic tenosynovitis usually occurs from an unresolved case of acute tenosynovitis; the tendon sheath is thickened due to continuous swelling. There may also be adhesion formation and a stricture of the tendon sheath that restricts movement and causes pain. Idiopathic tenosynovitis occurs for unknown reasons and causes swelling of the tendon sheath only. Infectious tenosynovitis is caused by infection and leads to pain, lameness, fever, swelling, and warmth.

Tendon Rupture

Tendons can rupture if they sustain enough force or if there is trauma done to a tendon which has previously been damaged. In the horse, the most commonly affected tendons include the gastrocnemius tendon, the peroneus tertius tendon, and the hind limb superficial digital flexor muscle tendons. When the gastrocnemius tendon ruptures, the hock joint becomes severely angled due to a dropped hock. A ruptured peroneus tertius can be diagnosed by extending the hock and flexing the stifle at the same time; in a normal horse, the stifle should extend. The fetlock joint is severely angled when a rupture of the superficial digital flexor tendon occurs. The rupture of this tendon causes the fetlock joint to drop towards the ground.

Perineal Laceration

Perineal lacerations occur while the mare is foaling. The foal's nose or foot catches on the vulvovaginal fold and tears the vaginal and perineal tissues. It is usually seen in primiparous mares, but also when there is forced extraction before the birth canal is fully dilated or if the foal is excessively large. There are three degrees of perineal laceration: A first-degree laceration is when only the mucosa of the vulva and vagina are lacerated. A second-degree perineal

laceration is defined as tearing of the submucosa and muscularis of the perineal body, anal sphincter, and vulva. A third-degree perineal laceration occurs when the laceration involves the rectovaginal septum, the perineal body, and the musculature of the vagina and the rectum.

A third-degree perineal laceration in the mare is a serious foaling injury. The injury is a tearing of the perineal body, the rectum, and the vagina and can result in conditions such as pneumovagina, endometritis, vaginitis, and cervicitis due to constant presence of fecal matter in the vagina. Before the horse can be bred, this wound needs to be repaired. The surgery should be postponed until the tissue swelling subsides and necrotic tissue is at a minimum. The mare should be carefully observed during this time period, as straining to defecate or urinate can result in prolapse of the intestines or bladder. The cervix can also be lacerated during the time of injury; this may result in difficulty in conceiving or carrying a fetus.

Inguinal Hernia

An inguinal hernia occurs when the herniated tissues pass through the vaginal ring into the inguinal canal. The distal jejunum and ileum are the most common organs to be herniated; less frequently, the omentum and small colon can herniate. These hernias affect male horses of any age, from the newborn to the adult stallion. In the newborn, the hernia is usually not strangulated and the typical sign is non-painful swelling. In the adult stallion, the hernia usually strangulates and results in painful swelling in the groin. Stallions usually develop a hernia after strenuous breeding, training, or racing. The horse develops colic-like symptoms; therefore, any stallion with colic should be rectally palpated to rule out an inguinal hernia. Rectal palpation will reveal a loop of intestine that has passed through the vaginal ring.

Contagious Equine Metritis

Contagious equine metritis is caused by the bacterium *Taylorella equigenitalis* , a venereally transmitted disease. The disease usually causes no clinical signs aside from fertility problems in the mare. Infected stallions also show no signs of disease. Contagious equine metritis currently does not occur in the USA. It was present in Kentucky in the 1970s, but was eradicated. Transmission is from stallion to mares during breeding, but it can also be spread by artificial insemination or through contaminated equipment. There are three categories of infection: A mare may be a carrier, may have an acute disease with uterine inflammation and passage of a thick discharge 10 to 14 days after being bred, or may have a chronic mild uterine inflammation. Treatment for both mares and stallions includes disinfecting the genitalia and the use of antibiotics.

Bone Spavin

Bone spavin is a condition of the hock joint involving the intertarsal, metatarsal, and intertarsal joints. It is an osteoarthritis causing lameness and may be due to conformational faults, mineral imbalances, excess protein or a protein deficiency, or excessive concussion. Bone spavin typically affects horses that race, jump, or are used for roping, cutting, and reining. A horse affected with bone spavin may be lame, have back pain, and suffer gait abnormalities such as dragging its toe. The lameness may disappear after exercise only to return when the horse rests. Radiographs may be used to confirm bone spavin; periosteal reaction is a typical sign. The joints may fuse after a few months, abolishing the cause of lameness.

Bovine

Teeth

Cattle are either born with or within two weeks develop four deciduous incisors along with three pairs of deciduous premolars. Within the first month of life, all eight deciduous incisors erupt. The incisors are only found on the lower jaw and meet with the dental pad on the upper jaw when eating forage. Therefore, there are usually twenty deciduous teeth in calves. At eight months of age, the first molars erupt. At one year of age, the first molars are fully developed, and at eighteen months, the second molars become fully developed. At two years, the third molar erupts and becomes fully developed at 30 months. The deciduous premolars are replaced by the permanent ones by three years of age. The central incisors are replaced between 18 and 24 months, the second ones at 24 to 30 months, the third pair at three years, and the fourth between 42 months and four years of age. The adult bovine usually has 32 permanent teeth by four years of age.

Respiratory Distress

Causes of respiratory distress in the ruminant can be caused by primary respiratory disease and nonrespiratory diseases affecting the entire body or specific organs. The respiratory diseases that commonly cause dyspnea include pneumonia, infectious bovine rhinotracheitis, necrotic laryngitis, and pulmonary edema and emphysema. Causes of pneumonia include lung worms, *Pasteurella* , bacterial pneumonia, aspiration or foreign body pneumonia, and atypical interstitial pneumonia. Other diseases can also have respiratory distress as a symptom; these include endotoxemia, anemia, anaphylaxis reactions, septicemia, and diseases affecting the abdomen, particularly those that put extreme pressure on the diaphragm such as bloat. Fever, electrolyte loss and disturbance, acidosis, painful conditions, fluid loss, and shock can also cause an animal to experience respiratory distress.

Respiratory Tract Disease

The bacteria implicated in ruminant respiratory tract disease include: *Actinomyces pyogenes, Bacteriodes* spp., *E. coli, Fusobacterium* spp., *Haemophilis somnus, Moraxella* spp., *Pasteurella* spp., *Peptococcus indolicus, Pseudomonas aeruginosa, Salmonella* spp., *Staphylococcus* spp., *Streptococcus* spp., *Anaerobes,* and *Facultative anaerobes* . Also, *Chlamydia* and *Mycoplasma* spp. can cause respiratory tract disease. Viruses that most commonly cause respiratory tract disease in bovines include: adenovirus, corona virus, calicivirus, herpesvirus (types 1, 3, and 4), influenza virus, enterovirus, bovine virus, diarrhea virus, parainfluenza virus type 3, reovirus, respiratory syncytial virus, and rhinovirus. Sheep and goats can have respiratory tract disease caused by these viruses: adenovirus, bluetongue virus, caprine-arthritis-encephalitis virus, parainfluenza virus type 3, respiratory syncytial virus, and bovine progressive pneumonia (maedi-visna) virus.

Review Video: Respiratory Disease
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Traumatic Injury to Pharynx

Traumatic injury to the bovine pharynx usually results from careless operation of calcium tubes, dosing syringes, stomach tubes, or baling guns. Other trauma can occur from stemmy roughage, foreign objects (metal fragments in hay or ground foods), indiscriminate ingestion of nail or wire, or puncture wounds over the pharynx. Any of these types of trauma can cause cellulitis or abscesses. The most common bacteria found in these wounds are *Actinobacillus* , *Actinomyces* , *Bordetella* , *Fusobacterium necrophorum* , *Pasteurella* , or *Streptococcus* . Prevention revolves around careful restraint and training in use of medication instruments, use of magnets in feed mills or grinders to keep metallic items out of the feed, pasture or barn cleaning and maintenance to remove loose wires and nails, and careful attention to proper roughage harvesting.

Smoke Inhalation

Smoke inhalation from barn fires may not be evident until 24 to 48 hours after initial insult. Damage to the respiratory tract can vary but includes inhalation of soot particles deep in the lung, edema, damage to the alveoli, necrosis, and secondary bronchopneumonia. Common problems include burns to the oral cavity, eye irritation, laryngospasm, hoarseness, rapid breathing, cough, and stridor. In addition to the assault by heat and toxic gases, carbon monoxide poisoning may occur, which starves the tissues of oxygen and decreases the

oxygen-carrying capacity of hemoglobin. Treatment involves keeping the air passages open, which may involve intubation or surgery to perform a tracheotomy. Supplemental oxygen along with bronchodilators and broad-spectrum antibiotics may be required.

Tuberculosis

Tuberculosis in the bovine is a serious disease with zoonotic potential. It is a progressive disease and causes localized granulomatous masses called tubercles. *Mycobacterium bovis* is the common cause of tuberculosis in cattle, although *Mycobacterium avium* and *Mycobacterium tuberculosis* do cause disease in cattle. The main reservoirs for *M. bovis* are humans and cattle. *M. bovis* is an acid-fast bacillus that enters the animal primarily through the respiratory tract. Its main focus is in the lungs, where it causes small, granulomatous lesions and eventually infiltrates into the lymph nodes. Bronchial, pharyngeal, mediastinal, and mesenteric lymph nodes can all be infected. From the lungs and lymph nodes other organ systems can be invaded. In ruminants, the disease is seldom overcome.

Tuberculosis caused by *Mycobacterium bovis* is a chronic debilitating disease with nonspecific signs related to the organ system(s) infected. One infected animal can spread the disease before it is realized that the organism is in a herd. Signs are commonly seen in the respiratory tract and are usually mild and may be confused with other diseases such as chronic pneumonia, mycotic pneumonia, abscesses caused by *Actinomyces pyogenes* , lymphosarcoma, botulism, laryngeal trauma or abscesses, or tumors. The only routine test is the interdermal tuberculin test, which can only be performed by an accredited veterinarian. Positive cases need to be reported to the appropriate authorities and the affected animals slaughtered. Necropsy findings typically reveal tuberculous granulomas in the lymph nodes, particularly ones associated with the lungs and digestive tracts, and the liver and lung tissue. The granulomas can have thick, yellow to orange pus or may be calcified.

The only test routinely used to identify animals infected with *Mycobacterium bovis* is the intradermal tuberculin test. Only accredited veterinarians can perform the test. 0.1 mL of mammalian tuberculin purified protein derivative (PPD) is injected intradermally in the caudal skin fold. After 72 hours (give or take six hours), the site is examined. Inflammation or swelling of the area indicates a suspect or positive test. Animals that do not exhibit these signs are considered negative. The most common test performed on positive or suspect animals is the comparative-cervical test. Federal veterinarians administer mammalian and avian tuberculin at the same time on the same side of the neck. After 72 hours, calipers are used to measure the reaction and the results are

compared with data on a chart to determine status. Positive animals are slaughtered and the herd of origin is depopulated in most cases.

Vesicular Stomatitis

Vesicular stomatitis is caused by a rhabdovirus that results in outbreaks of the disease in cattle and also in horses and pigs. It has a short incubation period (range three to 14 days) and is spread by poor milking equipment or hand sanitation. It is more common in older animals; cattle less than a year old typically do not show clinical signs. The signs of vesicular stomatitis are extremely similar to those of foot and mouth disease, and as such, it is a reportable disease. One distinguishing feature is that horses do not contract foot and mouth disease. Other diseases that can be confused with vesicular stomatitis include bovine viral diarrhea (BVD), infectious bovine rhinotracheitis (IBR), bovine papular stomatitis, irritation from bristle grass or toxins, and rinderpest. Signs include vesicles that rapidly coalesce into ulcerations, initial high temperature (greater than 106°F), decreased milk production, excessive salivation, anorexia with associated weight loss, and lameness (if the feet are involved).

Foot and Mouth Disease

Foot and mouth disease is caused by a picornavirus that attaches to the epithelium of the gastrointestinal tract. It causes vesicular lesions, ulcers, and erosions on the muzzle, in the mouth, on the teats, in the interdigital area, and on the coronary band. It is a highly communicable disease that is spread via aerosolization. Many animals can be infected, but cattle and pigs are the most susceptible to contracting the disease. Foot and mouth disease is a reportable disease that is not present in the USA. The clinical signs are identical to those of vesicular stomatitis. Other diseases that may be confused with foot and mouth disease include bovine viral diarrhea (BVD), rinderpest, bluetongue, infectious bovine rhinotracheitis (IBR), bovine papular stomatitis, and malignant catarrhal fever. Diagnosis is based upon clinical signs and laboratory identification of the virus. Treatment consists of premise quarantine and slaughter.

Cancer Eye

Cancer eye, or ocular squamous cell carcinoma, is the primary neoplasm of cattle. Herefords and other white-faced cattle are particularly susceptible to this lesion. Heredity along with long exposure to sunlight and dust are predisposing factors to acquiring this disease. The tumor can either be benign or malignant. Up to one third of the cases spontaneously regress. The tumor typically occurs on the conjunctiva, cornea, lower eyelid margin, or on the nictitating membrane. Other diseases that may be confused with cancer eye include pink eye, eye infections, ocular dermoids, other cancers, and trauma to

the eye or associated areas. The premalignant stage is typified by smooth, white plaques or papillomas. When the tumor is malignant, the edges become irregular, necrosis of the tissues may begin and emit a foul odor, and the tumor may spread into the bone or lymph nodes, particularly the parotid and cervical lymph nodes.

Diarrhea

There are many common causes of diarrhea in cattle. Parasites (worms) and coccidia cause numerous cases each year. Salmonella is the most common bacterial cause of diarrhea. Other diseases that cause adult cattle diarrhea are Johne's disease, colitis/enteritis, peritonitis, enterotoxemia, grain overload, bovine viral diarrhea (BVD), winter dysentery, malignant catarrh fever, and toxic plant ingestion. Systemic diseases such as septicemia/toxemia and heart, liver, and renal failure can also cause diarrhea. Mechanical problems of the digestive tract, such as a displaced abomasum, intussusceptions, cecal dilation, intestinal neoplasia, and vagal indigestion can cause loose, watery stools.

BVD

Bovine viral diarrhea (BVD) takes on many different forms of presentation; the majority of infected cattle show no clinical signs. In the classical form of the disease, signs related to the digestive tract predominate. These include explosive diarrhea, anorexia, depression, fever, rumen stasis with mild bloat, oral erosions, and dehydration. Most of these cases recover within 7 to 10 days. Others, especially those with more severe diarrhea and dehydration, have a fatal disease course. Mucosal disease is caused by chronic BVD infection and is invariably fatal within 2 months of exhibiting signs. Animals will have diarrhea, anorexia, will waste away, and have erosions on the nostrils, teats, vulva, coronary bands, and interdigital space. Some animals will have respiratory signs. Cattle can be difficult to breed and pregnant cattle can abort their calves.

Bovine viral diarrhea (BVD) infection during pregnancy causes a number of fetal problems depending on the stage of pregnancy at which the cow becomes infected. Early infection can cause early embryonic death or interfere with conception. Infection during the first 50 to 100 days can cause abortion or mummification of the fetus. Between 100 to 150 days, congenital anomalies may occur. These include cataracts, cerebellar hypoplasia, hydrocephalus, microphthalmia, or hypotrichosis. Some fetuses can become immunotolerant or become persistently infected; this generally occurs before the 125^{th} day of pregnancy. These fetuses can die in the uterus, may appear weak and die soon after birth, or appear to be normal; however, these normal-appearing calves can die if they contract the mucosal disease later in life. If infection occurs between

150 and 200 days of pregnancy, the fetus is considered to be immunocompetent, but can still die from infection or complications of viral replication in its organs.

Indigestion

Indigestion in cattle is a term used for a group of diseases caused by dysfunction of the reticulorumen. The most common cause is sudden feed change that disrupts the rumen microflora. Causes of primary indigestion include bloat (both frothy and free gas), diaphragmatic hernia, ruminal parakeratosis, obstructions, reticulitis/rumenitis, hardware disease, or vagal indigestion. Disruption of fermentation also causes indigestion. Disease conditions that disrupt rumen fermentation include lactic acidosis, rumen impaction or alkalosis, simple indigestion, and rumen ingesta putrefaction. In addition, there are a few diseases in which indigestion is a secondary symptom; these diseases include abomasal reflux and disruptions in reticulorumen activity or microflora.

Simple Indigestion

Simple indigestion in the bovine is a common condition. It generally occurs after an abrupt food change. It is caused when the rumen microflora are unable to adapt to the new feed, leading to an imbalance in the microflora and its fermentation products. The disease typically runs its course in 24 to 48 hours and consists of mild signs such as a slight decrease in rumen activity, mild bloat, and minor abdominal discomfort. The animal will go off feed for a day, have a bout of diarrhea for a day, and resume eating when the microflora adapts and the fermentation stabilizes. Feeds that may cause this condition include silages that are spoiled or partially fermented and overheated or moldy grains or hay. Indigestion will usually run its course without treatment, although some cases will benefit from rumen probiotics and antacids.

Grain Overload

Acute rumen lactic acidosis, or grain overload, occurs when the bovine consumes an excessive amount of carbohydrates. This produces rapid fermentation in the rumen, causing the rumen pH to drop to less than 5 due to the production of excess lactic acid. Grain overload occurs most commonly in feedlots when grain is introduced, if cattle are introduced to grain too quickly, or if cattle accidentally gain access to grain. It is more common in groups due to competition for feed among herd members. The decreased pH and the lactic acid disrupt the microflora and kill lactate-using organisms while increasing the number of lactate acid-producing organisms, such as *S. bovis* and Lactobacilli. These conditions lead to systemic acidosis, dehydration (fluid pulled into the rumen), and severe diarrhea. Hepatic, renal, and cardiac function are also impaired. It can be a rapidly fatal disease, with death occurring within a day of engorgement.

Diagnosis and treatment of grain overload depend upon clinical signs, history, and physical exam with associated laboratory results. Clinical signs generally appear from 24 to 36 hours after the engorgement occurs and depend upon amount of grain consumed and adaptation to a grain diet prior to consumption. Less severe signs are similar to those of simple indigestion: diarrhea, anorexia, bloat, a full rumen, abdominal discomfort, and reduced rumen motility. More severe signs are attributed to toxemic acidosis. These cattle become recumbent with their heads turned toward their flanks, are anorexic, have a subnormal temperature, severe diarrhea, dehydration, and little urine production, and have elevated heart and respiratory rates. Many of these animals die, especially if they are recumbent and/or exhibiting CNS signs (ataxia). Laboratory testing of the rumen fluid reveals a pH of less than 5, the blood pH is less than 7.2, and gram staining of microflora shows primarily gram-positive bacteria.

Free Gas Bloat

Free gas bloat (rumen tympany) is caused by physical interference with eructation. Typically an obstruction will occur at the cardia or in the esophagus. Passing a stomach tube will frequently relieve the gas and give the practitioner a diagnosis. Occasionally, the bovine will suffer from chronic free gas bloat. Calves with pneumonia that irritates the vagal trunks or cattle suffering from vagal indigestion may have this chronic form of free gas bloat. Signs are related to the amount of pressure the accumulated gas exerts on the body. Mild cases have the appearance of a mild distention in the left paralumbar fossa. More severe cases have greater distention, labored breathing, become anorexic, have excessive salivation and abdominal discomfort, and may become recumbent. These cases call for emergency relief of the bloat either through passing a tube into the rumen to relieve the gas or via a trocar into the rumen.

Frothy Bloat

Frothy bloat is caused by ingestion of pasture forage with high legume concentrations or through consumption of high concentrate diets. A foam forms in the rumen that traps the gas of fermentation. The bovine cannot dispel the gas via normal eructation. Signs are similar to free gas bloat, but there is a greater chance that an affected animal may be found dead before signs are noted. On postmortem examination, a “bloat line” may be noted. The cervical portion of the esophagus appears congested, while the thoracic part appears pale. The rumen contents appear to be of uniform consistency. Treatment of frothy bloat is via a rumenotomy to remove rumen contents and instill defoaming agents. Defoaming agents such as poloxalene or clothing detergent can also be used. Prevention hinges on pasture management and use of poloxalene in anticipation of frothy bloat.

Vagal Indigestion

Vagal indigestion encompasses a group of diseases in which the passage of ingesta from the reticulorumen or abomasum is impeded. In essence, the disease behaves as if the vagus nerve, which innervates the forestomachs and rumen, is cut. Vagal indigestion is usually observed in adult cattle. Some authorities have divided vagal indigestion into two types: Type 1 is considered to be an omasal transport failure in which the reticulo-omasal orifice becomes paralyzed and ingesta collects in the reticulo-rumen; the omasum and abomasum then remain empty. Type 1 is caused by a number of diseases: hardware disease, peritonitis, reticulitis, liver abscesses, physical obstruction of the orifice, or adhesions originating on the right medial wall. Type 2 is a pyloric outflow failure in which the pylorus becomes paralyzed. Ingesta accumulate in the forestomachs and does not pass into the small intestine. Causes of type 2 include abomasal volvulus, abomasal ulcers, displaced abomasum, or in late pregnancy.

Abomasal Obstruction

The primary cause of abomasal obstruction is poor quality roughage feed along with lack of water. This frequently occurs in pregnant beef cattle in cold weather. Other causes include calves with indiscriminate feeding (eating bedding or hair), lodging of ingested placenta, damage to the vagal nerve, surgery, or neoplasia. The bovine exhibits the "papple" shape in which the abdomen is pear-shaped on the right side and apple-shaped on the left side and begins to lose weight, have dry, firm feces in decreased amounts, goes off feed, and in severe or advanced cases, becomes recumbent. Diagnosis is generally made upon clinical signs and rectal palpation of a firm mass in the abomasum. Most cases will not be amendable to treatment due to the advanced stage. Other treatments, especially in early cases, depend on aggressive fluid therapy, laxatives, simple foods, and possibly termination of pregnancy in pregnant animals. Surgical treatment is generally unsuccessful.

Abomasal Ulcers

Abomasal ulcers, which are a full thickness erosion of the abomasal mucosa, are common in both adult and juvenile cattle. Causes may be varied, such as high-grain diets, parturition, lymphosarcoma, and viruses in adults. Calves may acquire abomasal ulcers by changing from a milk to grain diet, overgrowth of *Clostridium* , viral infection, hairballs, or copper deficiency. In adult cattle, ulcers can be staged according to type. Type 1 is a non-perforating and non-bleeding ulcer. Clinical signs are mild and include reluctant to feed and mild abdominal discomfort. Type 2 includes bleeding ulcers. The bovine becomes anorexic, milk production decreases, melena is noted in the manure, the animal shows

signs of colic, and heart rate increases. The ulcer may bleed enough such that it kills the animal. Type 3 includes ulcers that perforate, leading to local peritonitis that the body is able to "wall off"; signs include anorexia, decreased milk production, fever, and pain. Type 4 is similar but the peritonitis is diffuse and the animal dies of shock within a few hours of perforation.

Fatty Liver Syndrome

Cattle can suffer from fatty liver syndrome, which is more common in high-producing dairy cattle within the first month after delivery. Overfeeding during pregnancy leads to an obese cow and sudden anorexia after calving leads to a negative energy balance. Fat is mobilized from body deposits as lactation demands increase. The cow rapidly loses weight, fat becomes deposited in the liver (along with other organs), and the liver becomes dysfunctional. This dysfunction leads to hypoglycemia and an increase in ketone bodies and ketosis. The cow may become anorexia after delivery due to a number of factors, including mastitis, a retained placenta, hypocalcemia, bacterial infections (such as salmonellosis), metritis, or indigestion.

Ketosis

Ketosis is a metabolic disease of cattle primarily during the first six weeks of lactation. The primary causes of ketosis include obesity, inadequate dietary fiber, excessive silage consumption, lack of exercise, mineral deficiency, or lush pastures. Secondary ketosis develops when a cow is anorexic during early lactation due to disease. The pathophysiology of ketosis is as follows: The rumen microbes produce volatile fatty acids such as acetic, propionic, and butyric acid. Butyric acid is ketogenic, while propionic is a glucose precursor. The demands of lactation greatly decrease the liver stores of glucose such that fat is drawn from the body as an energy source. The fat is then metabolized and excess ketone bodies are spilled into the blood. Because the body is still deficient in glucose, especially the CNS, signs of ketosis soon develop in the cow.

Traumatic Reticuloperitonitis

Traumatic reticuloperitonitis, or hardware disease, frequently occurs in cattle. Dairy cattle are more frequently affected than beef cattle due to feeding practices. Cattle are indiscriminate feeders and will consume metal in the feed. These metal particles fall to the reticulum floor, and during contractions, the metal can be forced through the reticular wall, with the right medial wall being the site of most frequent penetration. Abscesses then form, most commonly towards the liver and occasionally towards the pericardium if the metal penetrates the diaphragm. This penetration also causes either localized or diffuse peritonitis, along with adhesions. When the metal penetrates the wall, the cow experiences pain, as indicated by reluctance to move, grunting, shallow

and fast breathing, and arched back, and goes off feed, and milk production will drastically cease. Chronically affected cattle exhibit signs of vagal indigestion, such as "papple" shaped abdomen, decreased manure production, and decreased rumen motility, as well as shock or fever.

Peritonitis

There are numerous causes of peritonitis in the bovine. Many times, the specific cause is not identified in the field. If the peritoneum is injured, it typically heals within one week, provided infection does not complicate healing. Normally, the peritoneum is a very permeable membrane and produces fluid to lubricate the abdominal organs. Fluid should be clear, with protein content approximately 3 g/dL; it contains fewer than 10,000 cells, some fibrinogen, and does clot when exposed to air. Most of the cells should be macrophages. Causes of peritonitis include hardware disease, wounds from the outside, vaginal tears, infected surgery, perforated ulcers, uterine rupture, abomasum rupture, bladder rupture, intestinal rupture, abscess rupture, fat necrosis, iatrogenic causes (irritating medications, perforation during AI, rectal tears during palpation), or a systemic infection.

Abomasal Displacement

Abomasal displacement is caused by high-concentrate diets along with infections such as metritis or mastitis. Feeding high-concentrate diets leads to an increase in volatile fatty acids, resulting in abomasal atony. The feed ferments, leading to gas accumulation; this in turn causes the abomasum to float up the side of the abdominal cavity. Abomasal displacement is more common early in lactation. Diagnosis is based upon physically examining the animal. Auscultation of the abdomen reveals gurgling sounds and percussion induces a pinging sound. In a left displaced abomasums, the "ping" is heard anywhere on the line from the tuber coxae to the elbow. With a right displaced abomasums, the ping is heard under the last five ribs. Occasionally, the right displaced abomasum twists, causing torsion, and the ping is also heard in the right paralumbar fossa.

RDA

A right displaced abomasum (RDA) and a right torsion of the abomasum (RTA) are considered to be surgical emergencies. A cow with an RDA will be off feed and milk production will decrease. A bovine with an RTA will appear very sick with an increased heart rate, dehydration, and no feces or small amount of watery manure, and is more likely than an animal with RDA or left displacement to die. Diagnosis is made through history and physical examination. Auscultation reveals a "ping" on the right side, occurring under the last five ribs in an RDA and into the paralumbar fossa in an RTA. Performing a rectal

examination allows the examiner to always palpate an RTA and occasionally an RDA. Laboratory testing is variable for an RDA, but an RTA produces abnormal values that reveal metabolic alkalosis, hypokalemia, and hypochloremia. Treatment is immediate surgery to correct the displacement.

LDA

Left-sided displacement of the abomasum (LDA) is more common than right-sided displacement. The cause of an LDA is not always known, but may result from a high-concentrate diet, along with infections of the uterus or udder or other stress. It is common postpartum. Some cases are difficult to diagnosis as the abomasum displaces and then returns to its normal position repeatedly. In a cow, signs include going off feed and decreased milk production, manure production, and rumen contractions. Percussion in a line from the tuber coxae to the elbow should reveal a high "ping," indicating gas accumulation. Treatment is surgery to place the abomasum in the correct position, although it is not considered to be an emergency.

Winter Dysentery

Winter dysentery in cattle is a contagious diarrhea that runs an acute course, running through a herd in less than two weeks, and typically occurring during the colder times of the year. Clinical signs include a watery, explosive, dark diarrhea (clotted blood), cough, decreased milk production, and anorexia. Some cattle become dehydrated and may have colic. The disease needs to be differentiated from gastroenteritis, coccidiosis, salmonella infection, Johne's disease, toxins, or bovine viral diarrhea. Treatment is usually not needed, as the disease is typically self-limiting, although owners should make sure cattle have adequate water and feed and possibly mineral supplementation due to ongoing losses from diarrhea. As the cause is not entirely certain, there is no vaccination against this disease, although a coronavirus is suspected.

Infectious Bovine Keratitis

Infectious bovine keratitis, or pinkeye, is caused by *Moraxella bovis,* a gram-negative coccobacillus. It is a rapidly contagious disease in some herds and affects calves more often than adults. Hereford cattle and their crosses seem more susceptible to the disease than other cattle breeds. There are many factors involved in the development of pinkeye. The organism itself has pilli, which bind to the corneal epithelium. Other contributing factors include exposure to ultraviolet light, heavy face fly load, dust, and concurrent infection with IBR or mycoplasma. Control of pinkeye involves controlling face flies through use of ear tags in the ears, insecticide dust bags, and fly-control lick blocks. Some cases are also treated with eye patches or subconjunctival injections of antibiotics.

Milk Fever

High-producing older dairy cattle five to nine years old are more likely to suffer from postparturient paresis or milk fever. Milk demands drain the calcium from the blood, causing hypocalcemia. Clinical signs depend upon the extent to which calcium levels are decreased. With levels around 6.5 mg/dL, the cow acts ataxic, vocalizes, or has muscle tremors. Calcium levels of 5.5 mg/dL cause a cow to become sternally recumbent; the cow appears sleepy, has dilated pupils, weak and rapid pulse, and no ruminal contractions, and typically the head is turned to the flank. With calcium levels below 5 mg/dL, the cow becomes comatose and predisposed to aspiration pneumonia, bloat, and death. Treatment is IV calcium gluconate given in a slow drip over a 20-minute period to prevent heart stoppage.

Poli Encephalomalacia

Poli encephalomalacia is caused by thiamine (vitamin B 1) deficiency and affects rapidly growing calves and lambs. Affected animals act as if they are star gazing, have dorsomedial strabismus, and appear blind with no menace reflex but intact papillary responses. Other CNS signs appear, such as head pressing, opisthotonus, ataxia, recumbency, and convulsions. Other signs include depression, anorexia, and decreased rumen activity. Causes of poli encephalomalacia include a high-carbohydrate diet with low roughage, which alters rumen microflora. Rumen microbes produce thiamine, and increased carbohydrates may change the flora to those that destroy thiamine, which is necessary to convert carbohydrates to glucose. In poli encephalomalacia, the brain is starved of glucose. Treatment is IV thiamine hydrochloride until the animal responds to treatment. Dexamethasone is also given to decrease inflammation and to stabilize membranes.

Anaplasmosis

Anaplasmosis is caused by the intracellular rickettsial organisms *Anaplasma marginale* or *Anaplasma ovis.* It is carried by ticks (*Dermacentor* and *Boophilus*), flies, and mosquitoes and can also be spread through dirty needles. Adult cattle are more likely to be severely affected than young stock. Affected adults have an acute hemolytic crisis; signs include weakness, pale mucous membranes, abortion, fever, going off feed, depression, decreased milk production, and possibly death. If it survives the acute stage, the animal loses weight and becomes icteric and dehydrated. It will take about one month for the animal to fully recover. Laboratory testing shows a regenerative anemia, low PCV (below 30 and possibly in the single digits), and an increase in unconjugated serum bilirubin. There is no blood in the urine, as the spleen

removes the diseased red blood cells. Giemsa staining will reveal the organisms in the blood smear.

Urolithiasis

Urolithiasis affects castrated male cattle. It is a metabolic disease caused by diet and environmental factors such as feeding on a high-concentrate diet, vitamin A deficiency, low water consumption, or high urinary pH levels. There are three syndromes associated with urolithiasis in the steer: The urethra can become blocked by a stone, the urethra can rupture from a stone obstruction, or the bladder can rupture. When the urethra is blocked, the steer at first dribbles bloody urine. After the blockage becomes complete, the steer develops anorexia, colic, and strains when trying to urinate, and the sheath becomes dry with crystals in the hairs surrounding the orifice. If the urethra ruptures, urine migrates into the subcutaneous tissue, leading to a "water belly" appearance. If the bladder ruptures, the steer eventually becomes very sick with peritonitis symptoms and eventually dies.

Meningitis

Meningitis caused by bacteria can occur either as the result of direct penetration of the skull or it can be caused by bacteria spread by the bloodstream. Common causes in calves include dehorning infection, sinus infection, ear infections, skull fractures, spinal infections, and septicemia resulting from an infection elsewhere in the body. The signs of meningitis begin with a fever, stiff neck, hyperesthesia, anorexia, and diarrhea. The animal may also display behavioral changes, such as lethargy or increased activity, convulsions, and coma. Diagnosis is based upon observation of the clinical signs and examination of the cerebrospinal fluid. The cerebrospinal fluid has white blood cell counts greater than 100 and protein concentrations between 20 and 270 mg/dL.

Urinary Tract Infections

Urinary tract infections of the bovine are usually caused by *Corynebacterium renale* or *E. coli* infections that ascend up the urinary tract. Conditions leading to infection include the presence of calculi in the urine, dystocia, metritis, conformational defects, iatrogenic causes, urine splashing, or venereal transfer of the causative agent. Clinical signs in the affected cow include dribbling urine, dysuria, pollakiuria, swishing the tail, blood or pus in the urine, scalding of the thighs or perineum, or in a bull, penis protrusion. If the animal has pyelonephritis, it may have generalized signs of anorexia, lethargy, colic, and fever and appear very ill. Dairy cows may abruptly stop producing milk. Physical examination may reveal a painful and thickened bladder, and if the infection is in the kidneys, enlarged and painful kidneys. Use of chemical urinalysis strips reveals hematuria and proteinuria.

Amyloidosis

Amyloidosis is the deposition of insoluble protein fibrils (amyloid) into various body organs such as the liver, digestive tract, kidney, and adrenal glands. This disease occurs sporadically in the bovine and is precipitated by chronic inflammation of other disease conditions. Amyloidosis is a disease of adult cattle and causes signs traced to the affected organs; generalized signs include weight loss, diarrhea, and decreased production. If the kidneys are affected, the amyloid is deposited in the glomeruli, causing excess protein to be lost in the urine, uremia with oral lesions, and generalized edema, which is particularly pronounced in the brisket and submandibular area. Other diseases to consider in a bovine presenting with this set of symptoms include Johne's disease, liver failure, lymphosarcoma, peritonitis, kidney failure from other causes, and parasitism. There is no treatment, and amyloidosis leads to death.

Leptospirosis Infection

When a cow is infected with Leptospirosis, the organism penetrates the mucous membranes and skin. It will then spread to body organs; antibodies stop the spread via the blood stream, but it persists in the kidneys, reproductive organs, eye, and cerebral spinal fluid. Most cases remain subclinical but other cases can cause an acute infection. Calves can have a hemolytic crisis, with fever, hemoglobinuria, hemolytic anemia, and icterus. Adult cows may have a condition called flabby udder mastitis when they will stop producing milk for 7 to 10 days. In addition, infection can cause abortions, especially during the third trimester, or produce weak calves. Treatment is with oxytetracycline or streptomycin in high doses. Vaccines are also widely available.

Horner's Syndrome

Signs of Horner's syndrome occur on the same side as the injury. Miosis occurs, which means the pupil appears small and contracted. Ptosis or dropping of the upper eyelid occurs due to paralysis of the periorbital muscles. The eyeball sinks into the socket (enophthalmos) due to periorbital muscle paralysis. The skin becomes hyperthermic and mucous membranes become congested. The horse displays sweating while the cow has loss of sweating on the nose (planum nasale). Treatment depends upon the underlying cause of Horner's syndrome. Unfortunately, once signs develop, there usually is little that can be done to reverse them. Occasionally, xylazine injections given intravenously cause signs of Horner's syndrome; this is usually a reversible condition.

Horner's syndrome is not a disease, but rather a sign of interruption of the sympathetic pathways in the head. The sympathetic pathway descends from the brainstem, down the neck where the nerves enter the gray matter at the level of T1-T3, synapse, and then exit through the ventral spinal nerves. The nerves

then pass through the cervicothoracic and middle cervical ganglia, pass up to the neck, and enter the cranial ganglion, where they synapse. From here, they are distributed to the sweat glands of the head, to the dilator muscle of the iris, to the periorbital smooth muscle, and to the periarteriolar muscles. These nerves can be damaged by many conditions, such as masses or abscesses in the thorax or cervical area, compression of spinal gray matter, injections into the neck, infections from the ear, or retrobulbar masses or abscesses.

Listeriosis

Listeria monocytogenes is the causative agent of listeriosis in cattle. It is a gram-positive rod found in the soil, feces, and also in silage, which is a common source of infection in cattle. Listeriosis causes brain stem meningoencephalitis, as well as septicemia in neonates and late-term abortions. It is usually a problem in individual animals as opposed to a wide-spread herd problem. Humans can get the disease from drinking milk from cattle infected with the bacteria or from contact with secretions or the products of abortion. The signs of listeriosis include neurological signs, such as circling, ataxia, anorexia, depression, head-pressing, dysphagia, conscious proprioceptive deficits, and fever. Animals with septicemia have fever, are depressed, and have a loss of appetite and diarrhea.

In animals with listeriosis, the cranial nerves (CN) V thorough XII are affected and cause the clinical signs. When CN V (trigeminal) is affected, the animal's jaw drops and it lose sensation in its face. Damage to CN VI produces medial strabismus in the cow. CN VII (facial) dysfunction causes the animal to have keratoconjunctivitis due to paralysis of the orbicularis oculi muscle, loss of menace and palpebral reflexes, a drooped ear, ptosis, and a drooped lip. Lesions on CV VIII (vestibulocochlear) cause head tilting, circling, ataxia, and nystagmus. When cranial nerves IX, X, and XII are damaged, the animal has difficulty swallowing, a paralyzed tongue, and stertorous breathing.

Downer Cow Syndrome

The downer cow syndrome occurs when a cow is unable to stand after being recumbent for greater than 24 hours. The primary cause of downer cow syndrome is the after-effects of parturient paresis. Other causes include dystocia leading to obturator nerve damage, coxofemoral luxation, fractures of the pelvis or pelvic limbs, spinal lymphosarcoma, injury to the spine, systemic illness (mastitis, peritonitis, metritis), metabolic disorders due to hypocalcemia (low phosphate, potassium, or magnesium levels), ketosis, liver failure, or muscle and nerve damage. Prolonged recumbency of cattle causes muscle ischemia and nerve damage within a matter of hours. After the initiating cause is corrected, the cow appear bright, alert, and responsive, attempts to creep or

crawl, eats and drinks, and passes normal manure. Other cows that are severely affected exhibit lateral recumbency and are in distress.

Mastitis

Mastitis in dairy cattle is a costly disease. There are four clinical presentations of mastitis. With peracute mastitis, the gland swells, feels warm, is painful, and produces abnormal secretions from the affected teats. The cow also becomes ill, with fever, depression, and sunken eyes, goes off feed and is weak. Acute mastitis has glandular changes similar to the peracute form but the cow does not appear as ill. With subacute mastitis, the animal does not appear to be sick, but changes, although not as dramatic, still occur in the affected glands. Subclinical mastitis is only detectable through tests such as the California Mastitis Test (CMT) or electronic cell counters. The CMT is done cowside; the reagents in the test kit react with the DNA of the white blood cells to give an estimate of the somatic cell count in the milk.

The most common causes of mastitis in cattle are *Staphylococcus* , *Streptococcus* , coliforms, *Pseudomonas aeruginosa* , and *Actinomyces pyogenes* . *Staphylococcus aureus* causes an acute and chronic form of mastitis that is difficult to eradicate. The most effective time to treat a cow affected with *S. aureus* is at the drying off stage. *Streptococcus agalactiae* is specific in that it needs the mammary gland to survive; because it is spread from cow to cow during milking, affected cattle should be milked last. Other *Streptococcus* spp. produce environmental types of mastitis. The most common coliforms that cause mastitis are *E. coli* , *Enterobacter aerogenes* , and *Klebsiella* . These pathogens can cause peracute or acute mastitis due to release of endotoxins. *Pseudomonas aeruginosa* causes persistent infection in cattle with subacute or acute flare-ups. *Actinomyces pyogenes* causes a foul-smelling purulent exudate, producing mastitis in dry cows and heifers; it is spread by the fly *Hydrotaea irritans* .

Brucellosis

Brucellosis in cattle causes a number of clinical signs. The first indication that a herd is infected with brucellosis is abortion of fetuses after the fifth month of pregnancy. Another sign is the birth of stillborn or weak calves. The infected cattle may also have reduced milk yields, retained placentas, mastitis, be lame, or may appear to be perfectly healthy. Bulls infected with brucellosis may have testicular abscesses. The reproductive organs, including the testicles, epididymides, seminal vesicles, and ampulla, as well as the semen, may be infected with *Brucella* organisms. Diagnosis of brucellosis is done via bacteriology by culturing aborted fetal tissue or uterine or mammary tissues or through serological testing.

Vibriosis

Campylobacter fetus sp. *venerealis* is an obligate parasite, or a gram-negative motile rod, of the bovine reproductive tract that causes the disease known as vibriosis. The disease is transmitted during breeding. Bulls carry the disease in the crypts of the prepuce. The disease primarily results in infertility or early embryonic death due to endometritis, which lasts three to four months. Infected cows become repeat breeders, with a high percentage of the infected herd returning to estrus. Cows may also have irregular estrus cycles. Another manifestation of vibriosis is abortion between five to eight months of gestation. The organism invades the placenta and the fetus, causing its death. The abortion rate in an infected herd is generally less than 10%.

Trichomoniasis

Tritrichomonas foetus is a flagellated protozoan that causes trichomoniasis in cattle. This organism lives in the genital tract, including the oviduct, uterus, vagina, and cervix; bulls serve as the mechanical vector for the organism. The organism does not interfere with conception; rather, it causes early embryonic death, infertility, and extended calving intervals. Sometimes the organism causes pyometra or retained placentas. If the cow is infected after the fourth month of pregnancy, the calf is usually delivered alive. Diagnosis is made by taking preputial scrapings from the bull or by culturing vaginal mucous, uterine exudates, placenta, or the contents of the fetal abomasum. Diamond's media is used to culture the samples obtained from the cow, bull, or fetus.

Anthrax

The organism responsible for anthrax, *Bacillus anthracis* , is a gram-positive, aerobic, capsulated non-motile spore-forming bacterium. The virulence of the organism is associated with two plasmids that code for toxin production and capsule production. *B. anthracis* produces an edema toxin and a lethal toxin that gain entry to the cells. The reticuloendothelial system and the vascular system are damaged by the bacterium. The organism thrives in alkaline soils and the spores are extremely resistant, living in the environment for decades. Cattle and sheep are the species most susceptible to anthrax, although other livestock such as horses and goats can be affected. Cattle and sheep are more likely to graze closer to the ground than horses and goats and therefore are more likely to ingest the spores.

Anthrax lives as spores in the soil. A typical outbreak in areas known to have contaminated anthrax soil occurs during the hot summer months (July, August, and September), especially after a dry period followed by a heavy rainfall or flooding. It is also more common when grasses become short and cattle are forced to graze "closer to the soil." The disease in ruminants occurs from one

day to two weeks after the cattle ingest the spores. Anthrax usually runs an acute course and most cattle are found dead. Those that exhibit clinical signs have a fever, bleeding from the body orifices, bloody diarrhea, blood in the urine, ataxia, convulsions, decreased milk production, and bloody milk. The animal may act excited or aggressive, then become depressed, have convulsions, and die. Chronic infections are extremely rare.

Diagnosis of anthrax in the ruminant is based upon clinical signs, a history of the disease in the area, and laboratory testing. It is extremely important that cattle that suddenly die in endemic areas and exhibit the clinical signs (especially bleeding from the body orifices) be handled carefully. In the dead carcass, the vegetative organisms become rapidly destroyed within a few hours after death. The soil becomes contaminated when these vegetative organisms are exposed to air (oxygen) and form the highly resistant spores. These animals should not be necropsied on the farm. Using a facial mask and gloves, a sample of blood from the jugular vein should be taken. Giesma staining will show a "string of pearls" of the bacterium. Other tests include polymerase chain reaction tests, fluorescent antibody testing, ELISA testing, or the Western blot test.

E. Coli

Enterotoxigenic *E. coli* causes diarrhea by producing an enterotoxin that affects the crypt epithelium of the jejunum and the ileum; this makes the intestines hypersecrete water and electrolytes into the intestinal lumen. Because young calves have receptors for the adhesins displayed by enterotoxigenic *E. coli* , a concurrent infection with rotavirus or cryptosporidia makes the animal more susceptible to the deleterious action of this bacteria. In addition, the enterotoxin produced causes further deterioration of the host: Diarrhea is non-bloody but very watery and causes minimal inflammation in the intestine. The calf quickly becomes dehydrated and may suffer from electrolyte imbalances; both processes may kill the calf if not corrected.

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Necropsy of a Cow

Before performing a necropsy on a cow, you should note the following: signalment, place of death, manner of death, external physical signs (blood in orifices), and whether any herdmates are affected with disease. The animal should be placed in right lateral recumbency. The first incision should be made midline on the skin on the lower jaw. Extend the incision along the body, coursing between the front legs and above the udder. Free the skin from the body wall on the upper side up to the spine if possible. Cut the right forelimb muscles between the shoulder and body. Reflect the leg back from the body. Lift the right hind limb and cut through the muscles toward the hip joint. Cut the ligament at the hip joint, and then reflect the leg back. Examine the udder and cut through all four quarters.

After the skin has been reflected off the right side of the cow, an incision should be made into the abdomen just behind the ribs; make certain you do not puncture the abdominal organs with this incision. Cut the abdominal muscles following the ribs. Cut the abdominal wall from the point of the sternum toward the udder, creating a flap of abdominal muscle. The abdominal muscles should now be exposed. Make note of the external condition of the abdominal organs and any fluid that escapes from the abdominal cavity. Cut and deflect the omentum from the abdominal cavity. The abdominal organs should now be sufficiently exposed for examination.

In cattle that have died of respiratory disease, lung tissue from both abnormal and unaffected lobes should be submitted to the laboratory in formalin and as fresh tissue. The liver, spleen, kidney, and heart should be submitted as fresh tissue samples. The formalin-preserved tissues can be checked for IBR, BVD, and BRSV by immunohistochemistry. The fresh tissues can be PCR tested for BVDV, IBRV, PI3V, BRSV, and Mycoplasma. In the pig, tissues that should be submitted in formalin include those of the lung (diseased and normal tissue), liver, spleen, kidney, and heart. Fresh tissue should be submitted from diseased and normal lung. In addition, a swab of the nasal turbinates should be submitted for culture, along with a cross-section of the snout if atrophic rhinitis is suspected.

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Ovine, Caprine and Cervidae

Border Disease

Border disease is a virus (Pestivirus) that causes abortions, mummification, or weak lambs in sheep. Other names of this disease are hairy shaker disease or fuzzy-lamb syndrome. Lambs that survive birth are persistently infected and spread the virus via secretions. The disease is first suspected when the flock begins to lamb. Fewer lambs than expected are born and the lambs that are born are small and hairy and may have tremors. The virus can be found in the blood and tissues of affected lambs by use of ELISA or PCR testing. There is no treatment or vaccination available. Sick lambs should not be used for breeding but it is helpful to expose un-bred ewes to these lambs to help develop immunity.

Ovine Progressive Pleuropneumonia

Ovine progressive pleuropneumonia (OPP) is a chronic, debilitating disease of sheep. The causative agent is the ovine lentivirus (a retrovirus also called Maedi-Visna virus), which is closely related to caprine arthritis encephalitis virus and is in the same subfamily as HIV and FIV. OPP is an interstitial pneumonia. Other disease conditions associated with this virus are polyarthritis, a neurological disease, and mastitis. The primary route of transmission is via lambs ingesting colostrum infected with the virus; a less efficient route is through ingestion of contaminated food or water or by inhalation of aerosolized virus. After infection, the virus replicates very slowly and the latent period may last from months to years. The viral genetic material becomes incorporated into the host's own DNA, leading to lifelong infection.

Bluetongue Disease

Bluetongue is indistinguishable from foot and mouth disease and vesicular stomatitis and is therefore a reportable disease in sheep. Arthropods, namely *Culicoides* or biting midges, transmit the virus, an orbivirus within the family Reoviridae. Two clinical forms of the disease occur in sheep: reproductive disorders or a type of vasculitis affecting several different organ systems. The vasculitis form causes such signs as fever, facial edema, salivation, nasal discharge, oral ulcers, hyperemia of the oral mucosa, pulmonary edema, secondary bronchopneumonia, lameness and stiffness, and death. Reproductive disease can manifest as dummy lamb births, abortions, or stillbirths. Prevention revolves around controlling the vectors of the disease (insects).

Pizzle Rot

Pizzle rot, or balanoposthitis, is fairly common in wethers but less so in rams. High feed protein levels, especially those caused by feeding on rich pastures, produce urine rich in urea. The alkalinity of the urine makes an ideal growth medium for bacteria, such as *Corynebacterium renale,* the primary culprit in pizzle rot, although other bacteria can cause the condition as well. The bacteria interact with the urea in the urine to produce ammonia. Strong ammonia can scald the prepuce and surrounding area, resulting in necrosis of the tissue. Scarring and stricture formation can block the urine flow and lead to retention of urine. Keeping the belly and prepuce closely shorn will help urine dry quickly. If the condition is caught early, removal of the animal from rich feed sources is successful at resolving the condition. Later stage treatment is rarely successful.

Orf or Soremouth

Orf or soremouth is a contagious ecthyma or contagious pustular dermatitis caused by a poxvirus of the parapoxvirus subgroup. The most common sign is shallow ulcers, which appear predominantly on the lips, in the nostrils, or on the feet. Young animals may be reluctant to nurse due to mouth and nose lesions; immunologically naïve older animals may also contract the disease. In the male, lesions also appear on the glans penis, preputial orifice, and prepuce while in the female, lesions will also appear on the vulva and teats. Orf is a zoonotic disease and therefore can be transmitted to humans. Orf is spread by direct contact, including sexual contact, although animals may be reluctant to mate due to the pain of the lesions. The disease typically runs its course in three to four weeks. Secondary bacterial infections are not uncommon, leading to more debilitating conditions.

Caseous Lymphadenitis

Corynebacterium pseudotuberculosis causes caseous lymphadenitis, which affects goats and sheep. The organism is a gram-positive rod that is passed into the environment and contaminates soil and manure. If an animal punctures itself with a contaminated object or brushes a wound against contaminated objects, the bacteria enter into the bloodstream or develop into an abscess at the area of entry; bacteria may also spread to the lymph nodes or to other internal organs and form abscesses. Typically, superficial abscesses full of thick, purulent material form that rupture and spread the bacteria to the environment and other animals. Abscesses can also cause problems with swallowing or breathing, interfere with the digestive processes, or cause arthritis, paralysis, or abortion, depending on location.

Porcine

Isospora Suis

Isospora suis is a parasite that causes neonatal piglet diarrhea. It is a coccidia consisting of round oocysts with a smooth single-layer capsule that contains two sporocysts with four sporozoites. *Isospora suis* is an intracellular parasite that develops in stages in the body and in the environment. The parasite lives in the pig's small intestine, specifically in the mucosa. Male cells then fertilize the female cells, producing an oocyst that passes from the body in manure; after one to three days, it develops into a sporulated oocyst. The oocyst is then eaten, passes to the small intestine, and releases the four sporozoites. These sporozoites enter the intestinal cells, divide, and reproduce; the offspring then invade and destroy other intestinal cells.

Trichurias Suis

The eggs of the parasite *Trichurias suis* can live for many years in contaminated soil. Infestation primarily occurs in recently weaned pigs that ingest the eggs; after ingestion, larva hatch in the small intestinal lumen and burrow into the wall. The larva then move on to the large intestine and cecum, where they mature into adults. The adult is shaped like a whip (whipworm) and is about 50 to 80 mm long; it begins to produce eggs in 41 to 45 days. With heavy infestation, the pig develops bloody diarrhea with mucous; it may also develop anorexia and depression. The disease can be confused with swine dysentery and colitis. Diagnosis is based on observation of the whipworm in the inflamed large intestine.

Vegetative Dermatosis

Vegetative dermatosis, or dermatosis vegetans, is a proliferative and inflammatory skin and hoof reaction that occurs in young pigs. A granulomatous alveolar and interstitial pneumonia may also be associated with

the skin condition. Vegetative dermatosis is a genetic disorder caused by an autosomal recessive gene of pigs of the Landrace breed. Newborn piglets have swelling and redness of the feet that spread up the legs to the abdomen. The lesions then become roughened, elevated plagues devoid of hair and are eventually covered with greasy, yellow-brown scales; the hoof walls develop ridges and become thickened. The pigs also acquire a mononuclear interstitial and alveolar pneumonia that progresses to a widespread granulomatous pulmonary infiltrate.

Causes of Diarrhea

Pigs suffer from a number of diseases in which diarrhea is the chief complaint. *E. coli* causes a severe watery diarrhea in neonates that persists through the post-weaning period. Salmonella also causes diarrhea and fever in pigs. *Clostridium perfringens* type C causes a hemorrhagic diarrhea in neonatal piglets, usually resulting in death. Another disease that causes hemorrhagic diarrhea in very young pigs is *Treponema hyodysenteriae* , or swine dysentery. Proliferative enteritis, also called terminal ileitis, causes mucous-laden and occasionally bloody diarrhea in growing pigs. The usual culprit in terminal ileitis is *Campylobacter sputorum* . Transmissible gastroenteritis is a disease that affects pigs of all ages but is especially devastating to piglets less than two weeks of age.

Actinobacillus Pleuropneumonia

Actinobacillus pleuropneumonia (APP) is a non-motile, gram-negative coccobacillus that causes respiratory disease in pigs throughout the world. It exists as two biovars: Biovar 1 contains 13 serovars and biovar 2 contains 2 serovars. The serovars exert different effects on the pigs and determine severity of disease. Biovar 1, serovar 1 is the most virulent strain. Growing pigs (three to four months of age) are most likely to contract the disease, but all ages are susceptible to APP. Transmission is direct or through aerosolization of virus. APP results in high mortality and represents a severe economic loss to swine producers. APP damages the lungs of growing pigs, leading to poor finishing weights; pigs may also become carriers, serving as a source for future infections. Outbreaks are seen during weather fluctuations or during severe winter weather.

Actinobacillus Pleuropneumonia

Diagnosis of *Actinobacillus pleuropneumonia* (APP) in swine is best obtained via necropsy. The lungs of affected pigs are darkened and consolidated and exhibit a fibrohemorrhagic, necrotizing pleuropneumonia; fibrin adheres to the surface of the lungs. In acutely affected pigs, the trachea and bronchi may be full of bloody foamy exudates, while chronically affected pigs may have pleural

adhesions and abscesses. Microscopic examination of the lung lesions shows suppurative, necrotizing severe fibrinohemorrhagic pneumonia. APP can also be grown on blood agar with NAD added for the growth of most serovars. Other tests that can be performed on live animals include ELISA, PCR (to determine serovar), ring precipitation, latex agglutination, immunoelectrophoresis, and immune-fluorescence.

Bacterial Diseases

Bacterial causes of disease in pigs include greasy pig disease, or exudative epidermitis, which is caused by *Staphylococcus hyicus* . This disease is most commonly seen in piglets less than two months of age and typically starts on the face and then travels down the body. There are two forms: the acute form, which manifests as a smelly, greasy, moist exudate, and the chronic form, which appears as scabby patches on the skin over the head and shoulders. *Borrelia suis* is a spirochete that causes lesions in young recently weaned pigs. This organism is found in unsanitary conditions and develops secondary to trauma from fighting; the ear becomes ulcerated and necrotic. Another condition that may follow attack of the ear by *Borellia suis* is porcine necrotic ear syndrome. *S. hyicus* and streptococci have been recovered from the lesions of affected pigs.

Dermatoses

Viral dermatoses can present as vesicular disease. Keep in mind that some exotic reportable diseases can be vesicular. Parvovirus can cause a vesicular dermatitis in piglets less than one month of age. Vesicles form on the tongue, snout, interdigital space, and coronary band. The pig can also have diarrhea, rhinitis, and conjunctivitis. Another disease presenting with vesicular lesions is swine pox. Swine pox generally does not cause death and is a fairly mild disease; clinical signs occur more commonly in pigs less than four months of age. The lesions are self-limiting and recovery takes less than a month. Swine pox is associated with unclean conditions; the swine louse, *Haematopinus suis* , is a probable vector of this virus.

Skin Lesions

There are a number of parasites that cause skin lesions in pigs. The one with the biggest economic impact is *Sarcoptes scabiei* var. *suis* , or sarcoptic mange. Sarcoptic mange affects pigs of all ages and the sow is the general reservoir in the herd. The mites live in tunnels they form in the pig's epidermis. Three weeks after acquiring mites, the pig develops lesions appearing as reddened, scaly patches with brown residue. The skin becomes intensely itchy and hair loss and skin abrasions form as a result of rubbing. Heavy infestations cause the pig to lose weight. Skin scraping is used for diagnosis; whole herd treatment with an acaricide will rid the herd of the infestation.

Infertility

A number of disease conditions cause infertility in the sow. *Brucella suis* or brucellosis is spread by breeding and causes embryonic death, abortion, and infertility, as well as orchitis in the boar. Porcine parvovirus causes infertility, mummification, and stillbirth in naïve herds. After herd infection, immunity to porcine parvovirus generally develops. Hog cholera causes a number of problems in the pregnant sow, including embryonic death, abortion, mummification, stillbirths, decreased litter size, and weak piglets. Pseudorabies in a pregnant sow causes abortion 10 to 20 days after signs of illness. *Leptospirosis pomona* causes abortions during the last month of pregnancy, although the sow may appear normal. The smedi virus causes many problems, such as abortions, mummification, stillbirths, infertility, weak piglets, and decreased litter size.

Pseudorabies

Pseudorabies (mad itch) is caused by a DNA herpesvirus that produces a highly fatal disease in swine and may also affect other animals. It is a reportable disease in the USA and has signs similar to those of rabies. Dogs and cats are considered dead-end hosts and die within days of acquiring the virus. The disease is spread by direct contact or indirectly through inhaling the virus. The virus can travel up to a mile and survives for days in certain conditions. It can be inactivated by high temperatures, drying, and sunlight. The virus replicates in the nose, tonsils, and pharynx of infected animals. Signs in neonatal pigs are attributed to the CNS, such as tremors and convulsions, and result in death. Older piglets show respiratory signs, such as anorexia, sneezing, fever, weight loss, or difficulty breathing.

<u>Feline and Canine</u>

Baylisascaris Procyonis

Baylisascaris procyonis is a common ascarid parasite of raccoons that infects dogs. The adult worms live in the raccoon's small intestine and eggs pass out of the raccoon in the feces. The single-cell eggs have a thick shell and under optimal conditions, embryonate in the soil to the infective stage. Raccoons ingest the eggs or the hosts that have ingested eggs and become infected. Dogs can either serve as hosts to the adult worm or behave as paratenic hosts and develop neurological signs, such as ataxia, blindness, paresis, or death. Canine adult worm infections can be treated with piperazine, ivermectin, fenbendazole, moxidectin, pyrantel, or albendazole. Dogs can potentially become carriers of this disease and pass it on to humans. This is a significant public health concern as *Baylisascaris procyonis* causes significant disease in humans that may result in permanent damage or death.

Giardia Felis

Giardia felis can be identified as a binucleate, flagellated trophozoite. The organism is found in the small intestine and moves down into the large intestine where it forms a resistant cyst wall. The cyst of *Giardia felis* divides in the large intestine, producing two trophozoites; this mature cyst then passes into the environment through the feces. Cats become infected by fecal-oral contamination and through contaminated water. A cat with an active *Giardia* infection will have clinical signs such as soft and pale diarrhea and will sometimes have weight loss. Kittens with *Giardia* will fail to gain weight despite normal appetite. Examination of a diarrhea sample using direct saline identifies the trophozoite. Centrifugal flotation with the zinc-sulfate method as well as antigen detection assays can be used to detect cysts.

Dirofilaria Immitis

The heartworm, *Dirofilaria immitis* , has a life cycle as short as six months. The adult *Dirofilaria immitis* live in the pulmonary arteries and right ventricles. Microfilariae are released into the circulatory system and are ingested by mosquitoes that feed on the infected animal. While in the mosquito, the L1 larvae migrate to the stomach and develop into L3 larvae, which then migrate to the mosquito's mouthparts. When the mosquito feeds, the L3 larvae are deposited in the bite wound and enter into the animal's bloodstream. Only 10 to 12 L3 larvae are transmitted to the animal by the infected mosquito. The L3 larvae molt to L4 and L5 (adult) larvae and arrive at the pulmonary arteries about three months after the animal is infected. After another two to three months, the adults sexually mature and migrate to the right ventricle.

Metabolic Acidosis

Metabolic acidosis is caused by the body producing excessive amounts of acid, or the kidneys not sufficiently removing acid from the body. Indications of metabolic acidosis include a decreased blood plasma concentration of bicarbonate and a high hydrogen ion concentration or a low blood pH. Metabolic acidosis may result from syndromes that cause an increase in acids containing chloride, such as diarrhea, addition of medications or fluids, or renal tubular acidosis. Another cause is the presence of anions other than chloride in the bloodstream, resulting from conditions such as diabetic ketoacidosis, ingestion of toxic substances, renal failure, lactic acidosis, and hyperphosphatemia. Animals with chronic conditions such as diabetes or hypoadrenocorticism or with renal failure are particularly prone to develop metabolic acidosis, as are animals with acute conditions such as toxin ingestion or diarrhea, or animals receiving carbonic anhydrase inhibitors.

Metabolic acidosis is diagnosed through blood serum chemistry testing. Calculating the anion gap may assist in diagnosing metabolic acidosis. The anion gap is calculated as AG= (Na +K)-(HCO3+Cl). A high or a normal anion gap will lead the clinician to suspect diseases such as hypoadrenocorticism, diarrhea (normal anion gap), diabetes ketoacidosis, kidney failure, and lactic acidosis; diarrhea is indicative of a normal anion gap. Depending on the underlying disease condition, the animal may also have high levels of glucose, lactate, phosphate, or potassium or may exhibit azotemia. To correct the disturbance, IV therapy consisting of lactated Ringer's solution with or without sodium bicarbonate, may be used.

ALT and AST

Elevated levels of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) on blood testing indicate that the liver has sustained some damage. These levels become increased due to a number of conditions, including those caused by chemical ingestion, viral, bacterial, fungal, parasitic, or protozoal disease, immune diseases, liver diseases such as hepatitis or cirrhosis, metabolic disturbances, cancer, digestive disorders, or endocrine diseases. ALT is considered to be a liver-specific enzyme although it can also be found in muscle and in the brain. AST is found in the liver, lungs, kidneys, brain, muscles, and erythrocytes; it is considered less specific to liver damage. In light of the fact that these enzymes can be found in other organ systems, high levels need to be evaluated along with other serum chemistry tests and whole blood cell counts in order to interpret the increased levels correctly.

Ascites

Ascites is a collection of serous fluid in the abdomen that may be either a transudate or a modified transudate. Transudate is a low-protein fluid that exits the intravascular space because of either increased hydrostatic or oncotic pressure. A modified transudate has a higher protein content, which usually occurs either because of hepatic lymph leaking into the transudate or because of inflammation. Causes of transudate include liver disease, protein-losing enteropathy, or kidney disease. Other causes of modified transudate include right-sided congestive heart failure, neoplasia, peritonitis, and in cats, lymphocytic/plasmacytic cholangiohepatitis. Occasionally, an effusion of high-protein fluid may be found due to serious damage to the biliary tract or urinary tract.

Diabetes Mellitus

Diabetes mellitus is caused by a deficiency in insulin; this deficiency can either be absolute (Type 1) or relative (Type 2). In type 1 diabetes, the pancreas stops producing insulin; in type 2, insulin is being produced but the body is not

responding adequately to the insulin. Insulin deficiency wreaks havoc on the body. The fatty tissues, liver, and muscle are unable to use nutrients properly, which impairs glucose production and use and leads to hyperglycemia; glucose then spills over into the urine, leading to glucosuria. The body tries to adapt by using fat stores for energy and produces ketones; this results in weight loss, fatty liver changes, and possibly ketoacidosis, a life-threatening condition.

A cat or dog with undiagnosed diabetes mellitus presents with a particular set of clinical signs. The owner will report that the animal drinks and urinates more, that it seems to be hungrier, and that it has lost weight. The animal may be overweight; a predisposition to developing diabetes mellitus. If the owner has not heeded these signs, the animal may be sicker when brought in for examination, and may be off feed, vomiting, and listless. Examination of the animal may reveal liver swelling (and also jaundice in cats) or trunk muscle wasting and an oily, dandruff- laden hair coat (especially common in cats). Dogs may suddenly develop cataracts, and cats with undiagnosed diabetes mellitus may develop diabetic neuropathy (a plantigrade stance). Blood testing reveals the following: high glucose levels, increases in ALT, AST, and SAP, increased cholesterol levels, and an increase in blood fat.

Ketoacidosis

Ketoacidosis is a life-threatening emergency in dogs and cats with poorly regulated diabetes mellitus. A lack of insulin causes an increase in fat mobilization from the body stores. The metabolism of fat causes ketone bodies to be formed and the blood to become acidotic. Because of ketone body production and acidosis, the body is unable to maintain fluid and electrolytes in the proper amounts; this causes azotemia, electrolyte problems, and dehydration. If not treated, the animal will die. Signs of ketoacidosis in a diabetic animal include increased drinking and urination, lack of appetite, listlessness, vomiting, dehydration, low body temperature, and weakness. Many diabetic animals with ketoacidosis either have poor regulation of their diabetes due to medication or diet problems, other health problems, such as kidney or heart disease or pancreatitis, or an ongoing infection.

Congestive Heart Failure

Congestive heart failure (CHF) can be attributed to failure of the left side or right side of the heart. In the dog and cat, left-sided failure causes blood to “pool” in the pulmonary circulation system or deprives the body of needed blood. Left sided congestive heart failure can result in disturbed heart rhythm and cause atrial tachycardia, ventricular tachycardia, or atrial fibrillation; these disturbances cause an increased heart rate, while an AV block causes a slower heart rate. The heart muscle itself may not function properly. The most

common cause of heart muscle failure is idiopathic dilated cardiomyopathy. High pressure on the heart/associated structures can cause left-sided congestive heart failure, as occurs with subaortic stenosis and hypertension. Other defects leading to left-sided CHF include pericarditis, endocarditis, mitral valve dysplasia, septal defects, cardiomyopathy, thromboembolism, and patent ductus arteriosus.

Congestive heart failure (CHF) can be caused by failure of the right side or left side of the heart. The right side of the heart can fail due to a number of reasons: parasitic load, structural defects, toxins, infections, or tumors. Heartworm disease, lung disease, stenosis of pulmonary veins, tumors, Tetralogy of Fallot, and pulmonary hypertension increase pressure on the right side of the heart. The heart muscle can fail due to idiopathic dilated cardiomyopathy or from doxorubicin toxicity. The right ventricle can be prevented from filling because of tricuspid stenosis, tumors, pericarditis, or pericardial effusion. Sometimes the heart rhythm may be disturbed (via AV blocks or supraventricular tachycardia), leading to right-sided congestive heart failure.

Review Video: Congestive Heart Failure
Visit mometrix.com/academy and enter code: 924118

Clostridium Perfringens

Both dogs and cats can have the syndrome known as clostridial enterotoxicosis. This large bowel diarrhea is caused by *Clostridium perfringens* , which releases an enterotoxin into the intestine that binds to the mucosa, causing cell damage and cell death. In the acute phase, the animal exhibits signs of large bowel diarrhea (frequent small amounts of bloody stool with mucus) that lasts for about one week. Other cases are chronic with intermittent periods of large bowel diarrhea. In addition to diarrhea, the animal may have an increase in gas, vomiting, abdominal pain, and generalized ill health. Treatment revolves around manipulating the diet to reduce clostridial numbers and acidifying the colon; this can be accomplished through a diet high in both soluble and insoluble fiber. Antibiotic therapy may also be indicated.

Coccidiosis Infection

Isospora canis causes coccidiosis in dogs, while *Isospora felis* causes the disease in cat. The organism sticks with its respective host. The organism causes dogs, cats, kittens, and puppies to have a loose, watery stool, possibly with mucus. In some cases, blood occurs in the stool as well. Puppies may become weakened. A fecal examination reveals the oocysts in the stool sample. Treatment choice is sulfadimethoxine (loading dose of 55 mg/kg by mouth for the first day, followed

by 27.5 mg/kg for four days). Other treatments include fenbendazole (50 mg/kg once daily for three days) or albendazole (25 mg/kg twice daily for two days).

Acute Diarrhea

There are many causes of acute diarrhea in dogs and cats. These can be grouped into broad categories such as parasitic-induced, drug- and toxin-induced, and infectious, dietary, and miscellaneous causes. Parasites implicated in causing acute diarrhea in the dog and cat are coccidia, ascarids, hookworms, Giardia, and Strongyloides. Infectious pathogens that cause acute diarrhea are *Rickettsia, Salmonella, Campylobacter, Bacillus piriformis, E. coli, Clostridial* spp*., Yersinia,* funguses, rotavirus, coronavirus, and parvovirus. Dietary causes of acute diarrhea include eating foods containing lactose or excess fat, abruptly switching foods, eating garbage or spoiled food, or overeating. Many drugs such as antibiotics and analgesics and toxins such as pesticides and insecticides may also cause acute episodes of diarrhea.

Syncope

Syncope is caused by the inadequate perfusion of the brain, which deprives it of oxygen and needed metabolic substrates. This leads to the temporary loss of consciousness and postural tone. There are a number of causes of syncope, including cardiovascular problems that can cause fainting. Other associated conditions include reduced cardiac output due to cardiomyopathy, infections, narrowed arteries, or veins in the heart or lung, as well as embolism, tumors, or heartworm disease. Nervous system or vasomotor problems such as emotional stress or carotid sinus hyperactivity can also cause syncope in animals. Drugs, hypoglycemia, hypocalcemia, or vascular (blood) disease can cause temporary loss of consciousness. Syncope should be differentiated from neuromuscular diseases such as myasthenia gravis or musculoskeletal disease that may cause an animal to collapse but not to lose consciousness.

Cough Reflex

A cough is caused when the vagus nerve endings in the larynx, trachea, and bronchi or when the glossopharyngeal nerves in the pharynx are stimulated. Coughs are divided into three broad categories: those caused by upper respiratory tract (URT) diseases, those caused by lower respiratory tract (LRT) diseases, and those caused by diseases of the esophagus or pleura. URT diseases that cause coughing include infections, tumors or foreign bodies in the nasal passages, pharynx, larynx, or trachea, or tracheal collapse. LRT diseases that cause an animal to cough include bronchial infections, inflammation, tumors, or allergies, lung infections, tumors, or edema, aspiration of foreign

bodies or substances, congestive heart failure, heartworm disease, pulmonary emboli or thrombi, or pulmonary hypertension.

Infective Endocarditis

Infective endocarditis is defined as an infection of the mural endocardium or of the heart valves. In order for infective endocarditis to develop, pathology of the valve or the endocardium must exist that allows bloodborne bacteria to invade and colonize the valves or endocardium. This infection then causes valve destruction and valvular insufficiency. The most commonly affected heart valves in the horse, dog, and very rarely, the cat, are the mitral valve and the aortic valve, while cattle are more commonly affected with endocarditis on the tricuspid valve. If the aortic valve is involved, left-sided congestive heart failure occurs within a few months. The signs of infective endocarditis include heart murmur, pyrexia, shifting leg lameness, exercise intolerance, lethargy, and cough and/or respiratory distress. If the aortic valve is involved, the examiner might detect hyperdynamic femoral arterial pulses.

Diagnosis and treatment of infective endocarditis can be difficult. Laboratory testing may reveal an active infection (left-shift leukogram, neutrophilia, and monocytosis), while those with a chronic infection may have a normal leukogram or one that shows a mature neutrophilia with monocytosis. Other test results might reveal thrombocytopenia, low albumin, low glucose, or high SAP activity. Because the kidneys are commonly affected by bacteria, urinalysis may reveal proteinuria, casts, hematuria, or pyuria indicating pyelonephritis or glomerulonephritis. Blood cultures should be performed to identify the bacteria responsible for the endocarditis. Echocardiography of the heart will readily reveal aortic valve endocarditis, and chest x-rays may show enlargement of the heart chambers or possibly a defective valve.

Skin Lesions

Skin lesions must be classified in order to accurately diagnose skin conditions. The common primary skin lesions and their typical appearance are as follows: A macule is a reddened or hyperpigmented discoloration of the skin that is less than one centimeter in diameter; a papule is a discrete, small, firm elevation of the skin surface; comedones are blackened blockages of follicles, similar to a human blackhead; a pustule is an elevated, reddened pus-filled bump; a nodule is a large (greater than one centimeter) solid lump elevated above the skin; erosions and ulcers are breaks in the skin with a slightly depressed area. An ulcer erodes the skin deeper than an ulcer directly to the basement membrane. A sinus is an infection that has eroded through the basement membrane.

Flea Bites

Flea bite hypersensitivity (or allergy) is commonly caused by *Ctenocephalides felis* (the cat flea) in both cats and dogs. It also causes the condition feline miliary dermatitis. The flea injects the dog or cat with saliva when it feeds on the host. This salvia, rich in many allergens such as amino acids, compounds similar to histamine, and polypeptides, induces hypersensitivity reactions (basophil, Type I, and Type IV). Dogs that have not been previously exposed to flea bites develop IgE and IgG antiflea antibodies. The dog can also develop either an immediate reaction and/or a delayed type of a reaction. It is not entirely understood how the cat responds immunologically to the bite of the flea, but most likely, it is in a manner similar to that of a dog.

A dog or cat with a true hypersensitivity reaction to a flea bite displays an itchy dermatitis. The cat may have a variety of clinical signs; the primary skin lesion is a papule that may take on a crusty appearance. The papules are found around the face, neck, and back; this pattern is called miliary dermatitis. The cat experiences intense itching, a rash, and possibly hair loss. In the dog, the allergic reaction causes an intense itching, often starting around the tail head or rear end. This reaction will cause the dog to constantly scratch, chew, and lick at its skin, which may result in hair loss, papules, skin discoloration, and secondary skin infections (hot spots).

Campylobacteriosis

Campylobacter jejuni causes the disease Campylobacteriosis, which affects dogs and cats, but is particularly prevalent and more severe in puppies less than six months old. The clinical signs of infection with *Campylobacter jejuni* include diarrhea (either acute or chronic), vomiting, regurgitation, pyrexia, loss of appetite, unkempt appearance, and loss of weight. In pregnant animals, abortions can occur if the fetus becomes infected in utero. The disease can mimic other intestinal diseases such as parasite infection, canine parvovirus, pancreatitis, or salmonellosis. To diagnosis the disease, a fecal smear can be examined under the microscope. This may demonstrate the darting, curved live causative agent. If the smear is gram-stained, the fixed organisms will be gram-negative rods that resemble gull wings. Cultures can be done on the fecal sample as well. Treatment can involve either supportive care alone or supportive care combined with antibiotics.

Heartworm

Heartworm disease is caused by the parasite *Dirofilaria immitis* ; the mosquito transmits the organism. The most common mosquito species that transmit *Dirofilaria immitis* are *Culex* , *Aedes* , and *Anopheles* , although many other species can carry the filarial worms. The most common companion animal to be infected with heartworm is the dog, although cats and ferrets can also become

infected. Outdoor dogs are more likely to be infected. The parasite causes cardiac and pulmonary lesions of variable severity depending upon the number of worms, how long the animal has been carrying worms, how active the dog is, and the immune response the dog has towards the worms. These factors can cause such conditions as congestive heart failure, exercise intolerance, vena caval syndrome, and glomerulonephritis.

Rabies

Clinical signs in a rabid dog or cat typically involve signs of central nervous system disturbance, primarily behavioral changes; these changes include nervousness, hyperexcitability, irritated attitude, and loss of appetite. A previously docile animal may suddenly become aggressive, or the animal may hide. There is generally what is termed a prodromal stage lasting from one to three days, during which the animal may have intense itching at the wound site and display temperament changes, apprehension, and possibly anxiety. The animal may then proceed to the furious form, with increased activity, biting, changes in the voice, salivation, ataxia, and eventually paralysis and death. More commonly, the animal may have the paralytic form, characterized by hiding, depression, muscle tremors, difficulty swallowing, and then death. Another form may develop in which the animal survives and becomes a carrier for a long period of time; this is called an unapparent form.

Diagnosis of rabies can be difficult because it has a variety of clinical signs upon presentation. History of exposure, bite wounds, and vaccination history are very important. The disease can mimic other causes of central nervous system disorders such as infections or toxins, canine distemper, listeriosis, pseudorabies, or feline infectious peritonitis. If there is human exposure, the animal should be quarantined for 10 days. If clinical signs develop in the animal during this time period, the animal should be euthanized and the head submitted to the laboratory for testing. The only definitive means to diagnose the disease is through immunofluorescence on brain tissue. Other testing methods that are not commonly used include mouse inoculation, immunoperoxidase staining, and polymerase chain reaction.

Nasal Polyps

Nasal polyps are pedunculated or elongated inflammatory growths that may resemble neoplasms. Usually, there is a single polyp in a nasal cavity. The outer surface is composed of smooth mucous membrane. The cat can have a very long solitary nasal polyp. The inner layers are composed of a mix of fibrous and myxomatous tissue. There is usually an infiltrate of granulocytes, lymphocytes, and capillaries. Epistaxis, or nosebleed, is usually caused by trauma to the nose or nasal passages, frequently by nasogastric tubes. Other causes include

ulcerated nasal tissues, infection of the sinuses or nasal passage, erosion of the nasal arteries from infection, neoplasia, or a fractured bone which has sliced an artery.

Glaucoma

Glaucoma is an optic disease that is caused by increased ocular pressure, leading to the loss of vision. It can develop as a primary condition from a genetic cause or it can develop secondary to other conditions such as retinal detachment, chronic eye problems, or cancer that develops in the eye. In most dogs, glaucoma develops because of genetic causes. Cats that develop glaucoma typically have another eye problem that leads to the development of glaucoma. Signs of glaucoma can occur in one eye or both eyes. Early signs include eye redness, a dilated pupil, and corneal edema. As the disease progresses, the eye may enlarge, the animal may be in pain, and the lens may luxate. Intraocular pressure increases from the normal range of 10 to 20 mm Hg to over 25 mmHg.

Primary or inherited glaucoma is caused when there is an increase in pressure in the eye because of abnormal draining of fluid through the iridocorrneal angle; this angle is used to classify the disease into either open angle or narrow angle glaucoma. If the angle where the iris and cornea join is wide, the animal has open angle glaucoma; however, if the iris base is pushed forward, it is known as narrow angle glaucoma. In the dog, narrow angle glaucoma is much more common than open angle glaucoma. Beagles have an inherited tendency to have open angle glaucoma. Open angle glaucoma is more likely to develop slowly over a matter of months; narrow angle glaucoma can cause a sudden increase in pressure in the eye. There are two other forms of glaucoma: goniodysgenesis, or presence of abnormal tissue at the angle, and pigmentary glaucoma, or presence of pigmentary cells blocking the angle.

Cataracts

Cataracts are defined as opacity of the lens or its capsule and are typically present in both eyes. The cat most commonly acquires cataracts as a secondary condition after chronic uveitis. Dog can develop cataracts because of genetic defects, as a complication of diabetes mellitus, because of trauma or neoplasia, or secondary to chronic uveitis. Cataracts are the most common cause of blindness in the dog, and usually affect dogs greater than five years of age. Some common dog breeds affected include Labrador retriever, cocker spaniel, Boston terrier, poodle, Bichon Frise, and miniature Schnauzer. Cataracts should be differentiated from nuclear sclerosis, which is a normal process of aging, or from lens imperfections in younger dogs. The only treatment for cataracts is lens removal.

Review Video: Glaucoma and Cataracts
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Retinal Detachment

Retinal detachment occurs when the retina is detached from the underlying tissues and becomes lifted or pulled away from its normal position. There are three different types of retinal detachment: Rhegmatogenous retinal detachment is caused by a tear or break in the retina, which allows fluid to flow under the retina and then separates the retina from the retinal pigment epithelium (RPE). The RPE is the pigmented cell layer that nourishes the retina. In tractional retinal detachment, scar tissue on the surface of the retina causes it to contract and separate from the RPE. Exudative retinal detachment is caused by an eye disease (inflammation or trauma) that causes fluid to leak underneath the retina without any breaks or tears in the retina.

Oral Tumors

Oral tumors in dogs can be benign or malignant. Cats typically do not have benign oral tumors. In dogs, the most common benign tumor is the epilus, or a tumor arising from the periodontal ligament (squamous cell epithelial cells); it is usually a solitary tumor around the molars (carnassials teeth). An epulis generally has a smooth surface and is closely adhered to the bone. Cats rarely have epulis; the most common benign mouth conditions in the cat are eosinophilic granulomas. The most common malignant mouth tumors in both the cat and dog are squamous cell carcinoma, malignant melanoma, and fibrosarcomas. Careful examination of the oral cavity during a routine physical examination is needed to catch these tumors in their early stages.

Fibrosarcomas are the second most common malignant oral tumor in the cat and the third most common malignant oral tumor in the dog. Fibrosarcoma is a locally aggressive tumor that invades the local bone but tends not to spread to lymph nodes or other body organs; it resembles a red growth on the gingival surface that may be necrotic or ulcerated. Larger breed male dogs over seven years of age have a slightly higher incidence of oral fibrosarcomas. Radiographs of the head will reveal bone lysis in the area of the tumor, but to definitively diagnose the condition, a deep biopsy must be submitted for histopathology. Treatment consists of a hemimandibulectomy with wide margins around the tumor.

Salivary Mucocele

A salivary mucocele occurs when saliva from a damaged salivary duct or gland leaks into the tissues around the duct. It usually presents as a soft, fluctuant

mass that is generally painless. A salivary mucocele causes an inflammatory reaction in the tissues and become lined with granulation tissue. There are four primary classifications of salivary mucoceles: sublingual mucoceles (ranula) occur alongside the tongue on the floor of the mouth, and cervical mucoceles occur underneath the jaw or in the upper neck region; this is the most common type of salivary mucocele. Pharyngeal mucoceles are collections of fluid in the pharynx. Zygomatic mucoceles occur just below the eye, with the saliva originating from the zygomatic salivary glands.

Feline Specific

Plague

Cats contract plague from eating dead rodents infected with *Yersinia pestis* , the causative agent of plague. This gram-negative bacterium causes an acute and sometimes deadly disease in rodents, rabbits, and humans as well. The flea transmits the bacteria through bites. Once the flea acquires the infection, the bacteria replicate in the flea's digestive system and multiply to such an extent that they block the flea's gastrointestinal tract, causing the flea to regurgitate the bacteria and spread it to the animal on which it is feeding. The disease can manifest in three forms: pneumonic, bubonic, or septicemia. After an animal is infected, the bacteria spread to the lymph nodes by the lymphatic vessels; the inoculated lymph nodes are then called buboes. If the organism spreads from the lymph nodes to other body organs, it is then called septicemia. Pneumonic plague develops either through septicemia or from inhaling aerosolized droplets from an infected animal.

Oral Squamous Cell Carcinoma

Oral squamous cell carcinoma is the most common tumor of the cat; it is a malignant tumor and invades the bone. The affected tissue appears irregular, is elevated, and is frequently necrotic and ulcerated. It is locally aggressive and typically does not spread to lymph nodes. In addition to a red, ulcerated spot on the gingival surface, the cat may have difficulty eating, excessive salivation, and bad breath, and may lose weight. The teeth surrounding the tumor may be loose as well. Biopsy is needed for diagnosis. Treatment involves radical surgery (partial mandibulectomy or maxillectomy) and radiation therapy. Frequently, the tumor will reoccur within a year, and the cat will die from anorexia and extreme weight loss.

Hyperthyroidism

Hyperthyroidism is a metabolic disease that affects middle-aged and older cats. It is the most common endocrine disorder in the cat. Enlarged nodules on the thyroid gland secrete thyroxine (T4) and triiodothyronine (T3). These enlarged nodules are usually benign tumors called adenomas; rarely is the disease caused by a malignant adenocarcinoma. Thyroid hormones affect most organs in the body. The heart can become enlarged. Hypertension can occur, causing damage to the heart, kidney, brain, and eyes. The gastrointestinal tract is affected as well, leading to malabsorption, diarrhea, and liver damage. The cat may have behavioral changes and appear anxious or have a ravenous appetite. All these effects are due to the increased metabolism caused by the high circulating levels of the thyroid hormones.

The clinical signs of a cat suffering from hyperthyroidism are attributed to the increase in metabolism created by high levels of thyroid hormone. The cat will lose weight despite a greatly increased appetite; it may have diarrhea, may become more thirsty than normal, have increased urine production, become hyperactive, and may vomit, and its coat may appear greasy and matted. Physical examination of a cat with hyperthyroidism will reveal an enlarged thyroid gland, heart murmur, increased heart rate, and poor body condition. Tests usually run to diagnose hyperthyroidism include complete blood count (CBC), blood chemistry, and a thyroid function tests. Most cats with hyperthyroidism will have an elevated level of T4 thyroid hormone.

Review Video: 7 Symptoms of Hyperthyroidism
Visit mometrix.com/academy and enter code: 923159

Otodectes Cynotis

Otodectes cynotis is a common mite that lives in the ear canal of cats. Kittens are commonly plagued by this mite, which causes irritation and a hypersensitivity reaction. Typical signs in a kitten include pruritis around the head and ears. The ears have a thick, dark, crusty material in the outer ear commonly extending into the ear canal itself. Many kittens will have scratch marks about the ear due to the intense itching. Diagnosis relies on ear swabs taken to remove exudates and then examining the exudates with mineral oil under the microscope to identify the mite. Treatment consists of thoroughly cleaning the exudates out of the ears, application of a topical product in the ear until cure is achieved, and treating the environment with pesticides. Alternatively, ivermectin may be used off-label.

Chlamydia Psittaci

Chlamydia (*Chlamydia psittaci*) causes a chronic respiratory infection in cats, with kittens aged two to six months most commonly affected. The bacterium multiplies in the upper and lower respiratory tract, causing signs referable to these areas. *C. psittaci* is also able to colonize the reproductive and gastrointestinal tracts as well. In the respiratory tract, the clinical signs seen in the cat include bronchitis, bronchiolitis, and rhinitis. All these symptoms are minor and cause coughing, sneezing, and watery eyes. Sometimes a cat will also get pneumonia from *C. psittaci.* Other disease conditions to consider when confronted with a cat with these signs are feline calicivirus (ulcerative stomatitis), feline viral rhinotracheitis (ulcerative keratitis), feline reovirus infection (very mild disease only in upper respiratory tract), or other bacterial causes of pneumonia. Tetracycline drugs are the agents of choice when treating a cat with Chlamydia.

Biliary Tract Disease

Cats can suffer from diseases of the biliary tract; specifically, cholangitis and cholangiohepatitis, or inflammation of the biliary ducts, the intrahepatic ducts, and the liver. There are three main types of disease: Lymphocytic and lymphocytic-plasmacytic types are caused by an immune reaction of the body; the suppurative type is most likely caused by a bacterial infection that may trigger an immune response and a lymphocytic and lymphocytic-plasmacytic reaction. Many cats with this inflammation of the biliary system and liver also have concurrent problems such as inflammatory bowel disease, cholecystitis, or pancreatitis. They may also have stasis of the bile, which may block the ducts or breed bacteria. Stasis is more common in middle-aged or older cats.

Chronic Diarrhea

Cats can have chronic diarrhea for many reasons, including parasites, fungus, inflammatory diseases, obstructions, infections, metabolic disorders, and dietary causes. Parasites that cause diarrhea in cats include *Toxoplasma gondii, Cryptosporidium, Dirofilaria immitis, Toxocara cati,* and Giardia. Fungus infections that cause chronic diarrhea in cats include aspergillosis, histoplasmosis, mycobacteria, and phycomycosis. Inflammatory conditions such as granulomatous enteritis, lymphoplasmacytic enterocolitis, and eosinophilic enteritis/hypereosinophilic syndrome can cause chronic diarrhea. Obstructions from cancer, foreign bodies, strictures, or an intussusception can lead to diarrhea in the cat. Many infectious agents cause chronic feline diarrhea. Viruses (feline leukemia virus, feline infectious peritonitis virus, and feline immunodeficiency virus) and bacteria (*Salmonella, Yersinia, Clostridium, and Campylobacter*) are major causes of diarrhea. Metabolic disorders such as

hyperthyroidism, diabetes, and kidney and liver disease have diarrhea as a symptom. Diets (sudden changes, intolerances, garbage eating) can also cause chronic diarrhea in cats.

Cryptococcosis

Cryptococcosis is a disease of cats that manifests as upper respiratory signs (rhinitis and nasal discharge), eye signs (retinal detachment, dilated pupils, optic nerve inflammation, or chorioretinitis), skin lesions, or neurological disease. The respiratory signs are more common in this disease, which is caused by inhalation of the fungus *Cryptococcus neoformans,* a yeastlike organism that tends to invade the upper respiratory tract (sinuses and nasal cavity). *Cryptococcus neoformans* is found in bird droppings, especially those of pigeons, and in soil. Given the right conditions the organism can live in the soil for up to two years. Treatment is with antifungals: itraconazole or fluconazole given in the food for one or two months after resolution of clinical signs.

Cytauxzoonosis

Cats can suffer from the disease Cytauxzoonosis, which is an infection with the protozoa *Cytauxzoon felis* . This rare disease is spread by tick bites (*Dermacentor variabilis*) and occurs in the bobcat and the Florida panther. The organism has an affinity for the vascular systems of many organs: brain, bone marrow, lungs, kidneys, liver, and spleen. After infection, within one to three weeks the cat will exhibit clinical signs. Cats contracting the protozoa will be severely ill with anemia, fever, dehydration, depression, enlarged spleen and liver, and icterus. The protozoa multiply and cause the body's macrophages to swell and to line the vascular system, which results in congestion in various organs. The disease is almost 100% fatal. Use of antiparasiticals to prevent tick bites may benefit cats in endemic areas.

Hypertrophic Cardiomyopathy

Cats can suffer from hypertrophic cardiomyopathy, which causes clinical signs such as respiratory distress, collapse, exercise intolerance, cough, or sudden death. These signs are attributed to the hypertrophy of the left ventricular wall or the interventricular septum. This enlargement of the left ventricle causes high filling pressures in this ventricle as well as arterial enlargement. The respiratory tract is also affected, leading to pulmonary hypertension and edema. Other problems include formation of thromboembolism due to blood stasis in the left atrium, heart arrhythmias, renal problems due to poor blood perfusion, and congestive heart failure. The heart, lung, and circulatory problems can cause signs in other body systems such as a weak femoral pulse; an aortic thromboembolism can cause sudden rear limb paralysis. If this occurs,

the animal will have cold limbs, no femoral pulse, and bluish nail beds and foot pads.

Ringworm

A sizeable number of cats suffer from ringworm (dermatophytosis). Dermatophytosis is caused by the fungi *Microsporum canis* , *Microsporum gypseum* , and *Trichophyton mentagrophytes* . The clinical signs of ringworm may include hair loss in a circular pattern, itching, redness, or scaling/flaking skin. Long-haired cats seem to be more likely to be infected. Cats can have the ringworm fungus but remain asymptomatic and can spread the disease to other animals and humans. The fungus lives in the superficial layers of the skin, in the hair and the nails. It grows in the keratinized layers after incubating for one to four weeks. Diagnosing ringworm relies on clinical examination combined with fungal culture.

Gingivitis/Stomatitis Syndrome

Feline gingivitis/stomatitis syndrome cause clinical signs of drooling, anorexia, halitosis, and oral pain. The physical examination reveals a cat suffering from gingival inflammation, stomatitis, pharyngitis, palatitis, and inflammation of the caudal fauca. Treatment of feline gingivitis/stomatitis syndrome begins with a thorough dental examination and prophylaxis. Teeth with end-stage periodontal disease and odontoclastic resorptive lesions should be removed. A biopsy of active lesions should also be taken and submitted for histopathological examination. Home care should include daily tooth brushing, chlorhexidine application, and antibiotic therapy using clindamycin, tetracycline, or metronidazole. Some cases are refractory to these therapies. Methylprednisolone acetate can be given every month to control the inflammation. In extreme cases, removal of all the teeth caudal to the canines can be attempted.

Oral Squamous Cell Carcinoma

The most common oral neoplasia of the cat is squamous cell carcinoma (SCC). The presenting clinical sign is most commonly an oral mass along with ptyalism; other signs include dysphagia, halitosis, and weight loss. Physical examination may reveal loose teeth, indicating bone lysis. This is an extremely aggressive and invasive malignant tumor. A deep incisional biopsy should be taken and submitted for histopathology. Chest x-rays should also be taken to check for pulmonary metastasis, and any enlarged lymph nodes should be aspirated for cytology. Other diagnostic testing such as CBC, chemistry, and urinalysis should be performed to assess health status. Treatment is not generally successful for this particular neoplasm, and in spite of treatment, most cases recur in six months.

Eosinophilic Granuloma Complex

The term eosinophilic granuloma complex is used to encompass three different syndromes in the cat: indolent ulcer, eosinophilic plaque, and eosinophilic granulomas. These conditions are characterized by an infiltrate of eosinophils into the epithelial tissue. All three may occur as a result of genetics or as an allergic, inflammatory condition and may come and go. An indolent ulcer is usually a raised area on the upper lips that tends to ulcerate. Eosinophilic plaques occur on the body, specifically, the armpit, the thighs, and the inguinal and perineal areas, and appear as hairless, reddened, moist patches. An eosinophilic granuloma can occur anywhere on the body. The lips and foot pad may swell and be painful and oral ulcerations may occur that cause drooling and difficulty eating. The body lesions may be white or yellow ulcerated areas; those on the caudal thigh may be linear granulomas.

Heartworm Infection

The cat has a different response to heartworm infection than the dog. Adult heartworms live in the cat from 18 to 24 months. The average worm burden is one to three worms; most cats do not have significant pulmonary hypertension or heart problems associated with this worm burden. Most of the symptoms displayed by an infected cat revolve around the lungs. The adult larva (L5) develops in the right and left pulmonary arteries and caudal lung lobes where most of the damage to the lungs occurs. In the cat, most of the L5 larva dies, and there is a strong inflammatory and eosinophilic response. As the worms grow, further damage occurs to the pulmonary arteries and the capillary beds. In response to this inflammation, the cat may exhibit asthma-like symptoms such as coughing and difficulty breathing.

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FIV

Feline immunodeficiency virus (FIV) is a lentivirus somewhat similar to the human immunodeficiency virus, although it does not infect humans. It is passed

from cats to cats primarily through bite wounds, as the virus is found in the saliva. Pregnant cats can transmit the disease virus to unborn kittens in the uterus or through the milk. Once infected, a cat is infected for its lifetime. An infected cat becomes very prone to infections via immune system suppression. After it is initially infected, there is a brief period when the cat's body responds with fever and swelling of the lymph nodes, along with neutropenia. The body then recovers and the cat appears normal for months to years before other signs appear.

The clinical signs of feline immunodeficiency virus (FIV) are extremely variable and the immune suppression can make the animal susceptible to many different disease conditions. The virus reproduces in the T-lymphocytes in the lymph nodes. The signs that the virus has compromised the cat's health include gingivitis and stomatitis, weight loss, unkempt hair coat, anorexia, and fever. The cat can also have skin infections, bladder infections, or upper respiratory tract infections; ocular inflammation and persistent diarrhea can be a problem as well. The disease can affect the neurological system and lead to behavioral changes or seizures. Arthritis, cancer, and anemia can also occur. The disease course can wax and wane, with periods of normal health interspersed with clinical signs.

Testing for feline immunodeficiency virus (FIV) antibodies is the primary means to diagnose the disease. If the test is positive, the cat is infected with FIV, as cats remain infected for life. Kittens less than six months of age that test positive should be retested, as kittens born to an infected mother can transmit her antibodies through the milk to the nursing kittens. Most kittens born to an infected mother will not be infected with the virus. It takes from two to three months after infection for the body to mount an antibody response. If a cat is bitten by another cat with an unknown FIV status, it should be test at least two months after the bite in order to determine if it is infected. Cats that have suffered from FIV for a long time may have a negative test result due to the severe immune compromise.

FIP

Feline infectious peritonitis (FIP) is a disease caused by a pathogenic strain of the feline coronavirus. The coronaviruses that affect cats are either the pathogenic strain or the feline enteric coronaviruses that do not cause disease or cause a very mild intestinal disease. If a cat is infected with the pathogenic strain of feline coronavirus, it will typically not display any symptoms or signs at the time it is infected other than an immune response that can be demonstrated by detecting antibodies in the blood. The clinical manifestation of FIP can take months to years to develop; however, only a small percentage (five to 10

percent) of cats that test positive will develop FIP. After clinical signs occur, death will ensue within a matter of weeks.

Feline infectious peritonitis (FIP) only occurs in a small percentage of cats infected with the coronavirus responsible for FIP. In these cats, the virus invades the white blood cells, with the assistance of the antibodies developed by the immune system in response to the viral infection. The infected cells then go to various body organs, and an inflammatory reaction occurs around the vessels where the cells have lodged; this typically occurs in the brain, kidney, or abdomen. FIP develops according to how the immune system responds to infection. There is a "wet" form caused by a weak cell-mediated response and a "dry" form caused by a moderate cell-mediated response. Carriers are either healthy or recover due to a strong cell-mediated response.

The signs of feline infectious peritonitis (FIP) vary depending on the form of disease. For either type (wet or dry), general symptoms include malaise, anorexia, fever, weight loss, and unkempt hair coat. The wet form causes a serofibrinous peritonitis or pleuritis due to vasculitis, which allows serum and protein to leak from vessels. It can also cause ascites and effusions around the heart. Due to fluid accumulation, the abdomen will enlarge. The dry form causes a granulomatous reaction involving the internal organs, eye, and/or brain. The dry form is so-called as there is no effusion present. The disease can be difficult to diagnose, although it should be strongly suspected if a cat has a fever that does not respond to antibiotic therapy. In addition, diagnostic tests are not specific enough to differentiate between the pathogenic coronavirus and the enteric coronavirus. Definitive diagnosis is made upon biopsy of necropsy samples.

FeLV

Feline leukemia virus (FeLV) is a retrovirus that causes a persistent infection in less than two percent of cats who contract the virus. There are three subtypes: A, B, and C; cats spread subtype A. The virus invades the tonsils and pharyngeal lymph nodes and infects B lymphocytes and macrophages, which spread the virus to various body tissues. In the cells, the retrovirus inserts copies of its genetic material into the host cells by use of reverse transcriptase. At the point of first infection, the immune systems of most cats eliminate the virus; a smaller portion will remain viremic for a period of time but eventually recover without any sign of infection. The two percent that are unable to mount an immune response remain viremic and may develop disease conditions associated with FeLV.

The signs of feline leukemia infection in the cat vary depending on the body tissues affected by the virus. It is the most common cause of cancer in the cat

and due to its attack on the immune system, leaves affected cats vulnerable to opportunistic infection by other viruses and bacteria that normally do not cause illness in cats. Some of the signs exhibited by infected cats include: gingivitis and stomatitis; recurring infections of the bladder, upper respiratory tract, and the skin; constant fever; anorexia; weight loss; generalized lymphadenopathy; unkempt appearance; constant diarrhea; signs attributable to the CNS; ocular infections or problems; and reproductive failure or abortion in intact females. The signs can wax and wane or they can lead to death.

A cat with clinical signs suggestive of FeLV requires a complete examination performed to rule out other disease conditions that mimic the various manifestations of FeLV. Diseases to include on the differential diagnosis list include feline infectious peritonitis, feline immunodeficiency disease, other cancer causes, haemobartonellosis, or bacterial, fungal, or viral diseases. An ELISA (enzyme-linked immunosorbent assay) test can be performed in the veterinary clinic that will detect primary and secondary stages of viremia. It is recommended that the test be repeated in three months to make sure the cat is persistently infected. At this point, an IFA (indirect immunofluorescent antibody assay) test can be performed to positively identify persistently infected cats. Both tests detect the p27 FeLV antigen.

Neoplastic FeLV

One of the disease manifestations of a cat persistently infected with feline leukemia virus (FeLV) is the neoplastic form. This is a lymphosarcoma that may develop in the gastrointestinal tract, the bone marrow, a generalized multicentric form, and cancer of the thymus. In the gastrointestinal tract, masses can be found in the intestines and mesenteric lymph nodes. The bone marrow (lymphoid tissues) produces cancerous lymphocytes that cause generalized lymph node enlargement and enlargement of the spleen and liver. Young cats frequently contract the multicentric form, which can cause kidney cancer along with enlargement of the liver and spleen. The thymus can become cancerous and enlarge, causing difficulty with breathing and eating. Another neoplastic form is myeloid leukemia, which affects various bone marrow cells, causing lymph node, liver, and spleen enlargement.

Non-Neoplastic FeLV

The non-neoplastic form of feline leukemia virus (FeLV) can cause immunosuppression, glomerulonephritis, or reproductive disorders. When FeLV causes immunosuppression in the cat, the cat may exhibit such recurring disease conditions as periodontal disease, upper respiratory tract infections, and infections around the claws; cats may also have poor wound healing and be chronically infected with any number of viruses, bacteria, or fungi. Female cats

with FeLV that are not spayed can have multiple problems with pregnancy, including abortions, stillbirths, or failure to maintain a pregnancy. Kittens born to FeLV-infected mothers may be weak and sickly and suffer from fading kitten syndrome. Another possible manifestation of FeLV is glomerulonephritis, in which antigen-antibody complexes are lodged in the kidney, leading to renal failure.

Feline Panleukopenia

Feline panleukopenia is an acute viral disease lasting for five to seven days. It is caused by the feline parvovirus a single-stranded DNA virus. The disease is highly contagious and the virus is highly stable in the environment and can last for years in favorable conditions. The virus spreads via infected secretions, urine, and feces and attacks the actively dividing cells in the blood and intestinal tract; this causes vomiting and diarrhea, leading to severe dehydration and lethargy. Most of the white blood cells in the body are lost; leukocyte counts typically drop from 500 to 3000 cells/microl. If a pregnant cat contracts feline panleukopenia, the virus will attack the fetal or neonatal nervous system. The virus destroys the cells of the cerebellum and retina.

Feline panleukopenia strikes suddenly in unvaccinated cats. Some cats will have mild symptoms; other will have a severe, sudden illness. The main symptoms are an extreme depression (sitting hunched up), hiding, dehydration, vomiting, diarrhea, nasal discharge, conjunctivitis, abdominal pain, and fever. Kittens can be infected in utero or soon after birth; they display central nervous system symptoms such as ataxia and gait abnormalities or problems with vision (retinal dysplasia). Other differential diagnoses include intestinal foreign bodies, feline infectious peritonitis, poisonings, herpesvirus infection, feline calicivirus, salmonellosis, or feline leukemia virus infection. Laboratory testing will reveal a severe panleukopenia (500 to 3000 cells/microl leukocytes). The feline panleukopenia virus antigen is detected on the canine parvovirus antigen fecal immunoassay. Serological testing on paired serum samples are also useful in arriving at a definitive diagnosis.

Periodontal Disease

Periodontal disease is a very common disease of the teeth affecting the majority of cats over the age of six years of age. It can be classified into five different stages of disease: At the beginning of the disease process (Stage 1), gingivitis occurs, which is caused by an accumulation of plaque; Stage 2 means the gingivitis is more advanced, but is still reversible. Stage 3 is the first stage of advanced periodontal disease, with gingival inflammation and deep pocket formation. During Stage 4, the pockets become deeper, there is loss of bone, and

teeth become loose. At Stage 5, the only treatment option is to remove teeth to relieve the pain, discomfort, and infection caused by neglected dental care.

Odontoclastic Resorptive Lesions

Feline odontoclastic resorptive lesions are common dental lesions that are very painful. The lesions include a loss of dental tissue at the neck of the teeth or the crown, various stages of resorption of dental tissue, and bright red gingival inflammation. Stimulation of these lesions elicits a painful response from the affected cat. In addition to the pain, the cat generally drools and does not want to eat. The cause is not known but it is suspected that the odontoclasts, which resorb tooth structure, become active; this activity cannot be prevented. The lesions have been classified into five stages: lesions only on the enamel, lesions that extend into the dentin, lesions that extend into the pulp, and lesions that cause crown destruction. Once the lesions extend into the dentin, it is recommended that teeth be extracted, as restoration attempts usually fail.

Canine Specific

Coccidioidomycosis

Coccidioidomycosis is a fungal infection found in dogs caused by the fungus *Coccidioides immitis* . Only a small amount of fungal spores are needed to cause disease and are typically introduced into the body through inhalation. The spores are found in arid regions of the southwestern United States in the lower Sonoran life zone (California through Texas). The fungus causes fever, weakness, lack of appetite, coughing, and listlessness. Occasionally, other body systems such as the joints may be painful and stiff. Treatment for this severe and life-threatening disease is long-term and aggressive use of an antifungal. Antifungals used in the treatment of coccidioidomycosis include ketoconazole (10-30 mg/kg three times daily) for one year or amphotericin B (.5 mg/kg IV every other day) until an accumulative dose of 8-10 mg/kg is given.

Chronic Diarrhea

Dogs with chronic diarrhea pose a diagnostic challenge. One of the first things the practitioner needs to do is localize the cause. This can be done by categorizing the diarrhea as small intestinal or large intestinal diarrhea. Based upon this characterization, different conditions can be investigated as the cause of the diarrhea. Small intestinal diarrhea is characterized by large-volume diarrhea, occasional vomiting, melena (but no frank blood or mucus), and weight loss. This type of diarrhea has a number of causes, including metabolic disorders, maldigestion, primary disease of the small intestine (within the intestine itself or due to parasites or infection), or diet. Large-bowel diarrhea is characterized by increased defecation frequency but in smaller amounts with

each defecation, blood and mucus in the stool, straining, and an urgent need to defecate. Causes of large-bowel diarrhea include dietary causes, metabolic disorders, intestinal problems (inflammation, cancer), parasites, or infection.

Cruciate Disease

Cruciate disease is a common surgical disease of dogs. The anterior cruciate ligament (ACL) can tear, either partially or fully, leading to lameness, pain, and arthritis. Animals that are obese, are a large breed, are vigorous athletes, or have a luxated patella or poor overall confirmation may be more prone to this injury. Cruciate disease may be caused by acute trauma or by a degeneration of the ligament. The ligament provides stability to the stifle joint by limiting rotation and displacement of the tibia and also functions to prevent hyperextension. The most common way the ligament tears is by traumatic forces that cause the stifle to hyperextend or by forceful rotation while the stifle is partially flexed.

ACL

Diagnosis of a torn anterior cruciate ligament (ACL) requires observing the clinical signs (lameness, pain, reluctance to walk), taking a thorough history of the inciting cause of the clinical signs, and obtaining a thorough physical examination. The most definitive way to diagnose a rupture or partial tear is through demonstrating a cranial drawer motion; this should be tested while the leg is extended, flexed, and at the normal standing position. Some animals, especially excitable, painful, or frightened dogs, will need to be sedated in order to get an accurate result. A negative result in light of clinical signs and history does not rule out a tear or rupture. Palpating the joint will demonstrate a joint effusion along with thickening of the joint capsule. Longer standing cases may have muscle atrophy.

Pododermatitis

Dogs suffering from pododermatitis (also known as interdigital dermatitis) can either have problems in one isolated spot on one foot or the problem can be widespread. In some cases, the cause is unknown (idiopathic). If only one foot is affected, the practitioner may suspect a neoplastic condition, imbedded foreign bodies such as plant awns, slivers of wood, or tree needles, or a possible case of osteomyelitis. If more than one foot is affected, the cause might be a systemic disease condition, such as a parasitic infection that also causes foot lesions (demodicosis, hookworm, or heartworm), a metabolic disease (hyperadrenocorticism or low thyroid levels), infections from fungus or bacteria, an allergic or sensitivity reaction, chemical burns, trauma, immune system problems (pemphigus, systemic lupus erythromatosis, or pemphigoid), or low levels of zinc.

Dermatomyositis

One breed-specific disease of dogs is dermatomyositis. This disease is an inherited condition that causes slight skin lesions and mild myositis to severe skin ulceration and scaring and muscle atrophy. It can be triggered by sunlight, stress, or factors related to the female reproductive cycle. Collies, Shetland sheepdogs, and their crosses are breeds affected by dermatomyositis. The lesions typically appear when the puppy is between two to six months of age. The dermatitis usually appears first on the bony prominences of the distal extremities, the tail tip, the face (eyes and lips), and the inner tips of the ears. Varying degrees of redness, scaling, hair loss, ulceration, and crusting occur in these areas. Severity of muscle inflammation may also vary from mild lameness to impairment of the muscles used for eating, drinking, and swallowing. Muscles on the face and jaw may even waste away.

Nasal Dermatoses

The causes of nasal dermatoses that a practitioner is likely to encounter in the canine include the following: demodicosis, contact dermatitis (from plastic food dishes), dermatophytosis, nasal pyoderma, discoid lupus erythematosus, dermatomyositis, systemic lupus erythematosus, squamous cell carcinoma, basal cell carcinoma, fibrosarcoma, trauma, zinc-responsive dermatosis, pemphigus foliaceus, pemphigus erythematosus, drug sensitivity, and granulomas. Some specific breed predilections for nasal dermatitis include Alaska malamutes; dogs under a year old and Siberian huskies are prone to zinc-responsive dermatosis. Young dogs less than a year old are more like to suffer from demodicosis and dermatophytosis. Dermatomyositis is more likely to be found in young dogs, collies, and Shetland sheep dogs. Collies, German shepherds, and Shetland sheep dogs are predisposed to discoid lupus erythematosus and systemic lupus erythematosus.

Atopy

Atopy is a common condition in the dog that usually develops in the first years of life; this is the tendency for the dog's body to develop skin hypersensitivity to environmental allergens. Allergens commonly implicated in canine atopy include molds, pollens (weed, grass, and tree), dust mites, and animal dander. The typical reaction includes exposure to the allergen, which is associated with an IgE. After repeated exposure to the allergen, its IgE then binds to skin mast cells, which degranulate to release compounds causing intense itching. The hypersensitivity reaction can be year-round or seasonal depending on the allergen cause. As the years progress, atopy becomes worse and a seasonally dependent allergy may become a year-round allergy. Differentials to atopy

include flea bite hypersensitivity, food allergy, contact dermatitis, yeast allergy/infection, or mange.

Babesiosis

Babesia gibsoni, a protozoan, causes babesiosis, a severe red blood cell disease in dogs. Another type, *Babesia canis* , causes a mild disease in adult dogs but can kill exposed puppies. In dogs with Babesiosis causing red blood cell destruction and anemia, the clinical signs include anorexia, pale mucous membranes, listlessness, fever, vomiting, discolored urine, increased heart and respiration rate, enlarged spleen, and icterus, all generalized signs of anemia. Blood work reveals a hemolytic anemia, as indicated by spherocytes, anisocytosis, and polychromasia. Diagnosis is based upon demonstrating the organism *Babesia* in the blood; however, these are sometimes difficult to find. Other tests to help diagnosis the condition include titers, Coombs' testing, and indirect fluorescent antibody tests.

Blastomycosis

Blastomyces dermatitidis causes blastomycosis, a systemic fungal infection affecting the lungs, male genitalia, eyes, and/or skin of dogs. Hunting or sporting breeds of dogs are especially prone to acquiring this fungus, as they are more exposed to environments harboring *Blastomyces* through inhaling the fungus or through skin wounds. Presentation of clinical signs depends upon the area affected. Skin lesions manifest as abscesses or thickened areas that ooze fluid and are ulcerated. If bone is invaded from the skin, an osteomyelitis will occur; if this occurs on the leg, the dog will be lame. Some male dogs have lesions such as skin lesions in the testicles or have an enlarged prostate. Ocular infection presents as a uveitis. In the respiratory tract form, a dog presents with difficulty breathing, fever, cough, enlarged lymph nodes, pneumonia, and weight loss.

Bordetella Bronchiseptica

Kennel cough or infectious tracheobronchitis is caused by *Bordetella bronchiseptica* and is a major respiratory pathogen of dogs. It is a gram-negative bacterium that is transmitted through the air and through contaminated fomites. Bacteria colonize the respiratory tract epithelial cells and excrete endotoxins and exotoxins that harm the respiratory tract and suppress the immune system. The primary clinical sign is a paroxysmal hacking, dry cough that becomes worse with exercise or palpation of the throat. Generally, the disease is self-limiting, although antibiotics and other medications are used to ease symptoms. Antibiotics most effective against *Bordetella* include erythromycin, azithromycin, tetracycline, clarithromycin, and chloramphenicol. Other antibiotics include trimethoprim-sulfamethoxazole and cephalosporins.

To control coughing, limit the dog's exercise and use bronchodilators and antitussives. Vaccinations (modified live, whole cell bacterin, extracted cellular antigens) are available in either intranasal or injectable form.

Distemper

Canine distemper is diagnosed based on clinical signs and laboratory testing. Clinical signs are variable, from no visible signs to a severe disease with central nervous system signs. A diphasic fever occurs; the first three to six days post-infection and the second several days later, along with eye and nose discharges. Generalized signs include anorexia and listlessness. Some dogs experience secondary gastrointestinal, ocular, and respiratory signs as well. The CNS component manifests as seizures, myoclonus, ataxia, paralysis, muscle tremors, and paresis. Laboratory work-up reveals lymphopenia and thrombocytopenia during the early acute phase; monocyte numbers may increase. Other tests to pinpoint the virus include ELISA and PCR. Dogs with CNS signs may have increased mononuclear cells and protein in the cerebrospinal fluid.

Causes of Abortion

The causes of abortion in the dog can be categorized as follows: fetal defects, reproductive organ dysfunction, infectious organisms, and hormonal dysfunction. Fetal defects that cause a bitch to abort are chromosomal defects inhibiting life and fetal organ defects. Problems with the bitch's reproductive organs may also interfere with carrying a litter, including lysis of the corpora lutea associated with use of prostaglandins, estrogens, high dosages of glucocorticoids, or drugs toxic to the embryos, as well as trauma, neoplasia, use of chloramphenicol, pyometra, and cystic endometrial hyperplasia. A number of infectious agents are abortogenic, including viruses such as distemper, parvovirus, and herpesvirus. Bacteria that cause canine abortions include *E. coli* , *Campylobacter* , *Streptococci* , and *Brucella canis* . Other organisms that cause abortion in the dog are toxoplasma, Ureaplasma, and mycoplasma. Hormones can also cause abortion in the dog. A dog with uncorrected hypothyroidism or with hypoluteoidism can abort a litter.

Diaphragmatic Hernia

A diaphragmatic hernia is a surgical emergency. In dogs, diaphragmatic hernia may result from congenital defects, or more commonly, from trauma. Being hit by an automobile is the primary traumatic cause of the hernia. The diaphragm muscle tears or ruptures, leading to the intestinal organs bulging through the opening and into the chest cavity. Hypoventilation results from the lungs not being in normal contact with the parietal pleura or due to fractured ribs. Bulging of the abdominal organs and fluid accumulation also contribute to hypoventilation. In addition, the lungs may collapse or be bruised. The heart

muscle may also be traumatized, leading to cardiac problems. As a result of pain, trauma, and lung issues, the animal typically goes into shock and may die before surgical intervention. Physical signs include respiratory distress, muffled heart sounds, and shock.

Otitis Externa

Otitis externa frequently occurs in the dog. It is an inflammation of the skin lining the external ear canal and can be either acute or chronic. The ear becomes reddened, there is an increase in exudate from the ear, and the dog's skin is pruritic and/or painful. Some dogs are prone to developing ear infections due to poor ear canal conformation or excessive hair in the ear canal. Bacteria (staphylococci or streptococci) or fungus (*Malassezia pachydermatis*) are common causes of otitis externa infection. The ear canal should be thoroughly examined to assess the degree of infection and to make sure the tympanic membrane is intact, and sedation should be used if the ear is painful or the dog is uncooperative. Therapy should only be initiated once the cause of infection is determined. Typically, ears should be cleaned of exudate before medications are placed in the ear canal.

Ectropion

Ectropion is a condition of the eyelids in which the eyelid margins either roll in or out; this causes the underlying conjunctiva to become exposed and causes problems with tearing. The dog presents to the veterinarian with eyelid redness, thick discharge from the eye, and often secondary bacterial infections or even corneal ulceration. This condition may be due to genetics; certain breeds are predisposed to developing ectropion such as bloodhounds, the St. Bernard, and the Mastiff. Other causes include hypothyroidism, nerve paralysis, or myositis. In animals with minimal symptoms, medical care including facial cleansing, treatment of any underlying medical conditions, eye medication, and observation may be all that is needed. Other more severe cases may need surgery such as eyelid shortening or a face lift procedure in order to prevent loss of vision.

Hypoadrenocorticism

Hypoadrenocorticism, otherwise known as Addison's disease, is a fairly rare disease found primarily in young, female dogs. Primary hypoadrenocorticism is most likely caused by an autoimmune destruction of the adrenal cortex. This destruction results in the body becoming deficient in cortisol (glucocorticosteroid) and aldosterone (mineralocorticoid). Cortisol is released from the adrenal glands in response to ACTH produced by the pituitary gland. It is necessary in a number of important bodily functions, including glucose metabolism, regulation of blood pressure, immune function, and insulin release,

and aids in the inflammatory response. Aldosterone works in the kidney to increase the reabsorption of sodium and water and to release potassium. These actions help the body to maintain a constant blood pressure.

Hypoadrenocorticism (Addison's disease) may cause non-specific signs. A typical sufferer is a young to middle-aged female dog. Some breeds that appear to have a greater risk of developing hypoadrenocorticism are Portuguese water dogs, poodles, Rottweilers, Great Danes, West Highland white terriers, and Wheaton terriers. The dog may have a poor response to stress, episodes of gastroenteritis that come and go, or a fulminant gastroenteritis that leads to collapse (a hypoadrenocortical crisis), as well as a loss of body conditioning. Other signs include lack of appetite, increased drinking and urination, dehydration, hyperpigmentation of the skin, depression, vomiting, diarrhea, abdominal pain, and/or weakness. Laboratory diagnosis reveals increased potassium and calcium levels, low levels of glucose and sodium, azotemia, anemia, eosinophilia, and lymphocytosis. An ACTH stimulation test will help to definitively diagnosis Addison's disease.

Review Video: Addison's Disease
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Megaesophagus

Megaesophagus, or dilation of the esophagus, can be either a congenital problem or can be acquired later in life. Animals may be born with esophageal diverticula or with vascular ring anomalies or the dilated esophagus may be idiopathic. In adult animals, megaesophagus may have an idiopathic cause or it can develop secondary to other diseases. The diseases that can cause megaesophagus include esophageal lesions, nervous system disorders, myasthenia gravis, polymyositis, hypoadrenocorticism, systemic lupus erythematosus, dysautonomia, hypothyroidism, or lead poisoning. The primary sign of megaesophagus is regurgitation. Puppies with the disorder will typically be smaller than littermates and begin to regurgitate as soon as they begin to eat solid food. Adult animals also regurgitate and start to lose weight. Both adults and puppies will have respiratory problems due to aspiration of food.

Hemangiosarcoma

Canine hemangiosarcoma is a very malignant tumor originating in the endothelial cells. Dogs are more likely to suffer from this condition than other animal species, and large breed dogs, particularly Golden retrievers and German shepherds, over the age of 10 years are more likely to succumb to the disease. It is not known what the inciting cause of hemangiosarcoma is in the dog,

although chronic ultraviolet irradiation is a risk factor for superficial dermal hemangiosarcoma in certain breeds, such as short-haired lightly pigmented dogs. While the prognosis for hemangiosarcoma is grim, standard therapy for canine hemangiosarcoma involves surgery to remove or debulk the tumor, chemotherapy with doxorubicin (with or without use of cyclophosphamide or vincristine), and radiation therapy.

Tracheal Collapse

Dogs that suffer from tracheal collapse quite frequently also have chronic bronchitis. For a dog to be diagnosed with chronic bronchitis, it must have a chronic cough lasting at least two months that is not caused by some other anomaly or infection. When a dog has a collapsing trachea, it has an excessive instability of the trachea, which may also be paired with collapse of the large bronchi. A dog suffering from tracheal collapse will give a characteristic "goose honk" cough when the trachea is palpated, when there is pressure from pulling on a lead and collar, or when the trachea collapses in on itself. This problem is more frequently reported in small breeds that are middle-aged or older.

Diagnosis of tracheal collapse begins with history-taking that reveals a long-term chronic cough made worse by pressure around the trachea. The physical exam should pay particular attention to the cardiopulmonary system. Other conditions such as congestive heart failure, tumors, primary lung disease, or infections can also cause long-term coughing in dogs. Because Cushing syndrome is found quite frequently in dogs with the same history, it may need to be ruled out as well. X-rays of the chest are helpful in the diagnosis. Many dogs with chronic bronchitis have an increased bronchial pattern. When a dog with tracheal collapse is radiographed, cervical tracheal collapse can be seen during inspiration, while x-rays during expiration can expose an intrathoracic tracheal collapse. Bronchoscopy can be used to visualize the trachea and bronchi and to collect samples for cytology and culturing.

Exocrine Pancreatic Insufficiency

Exocrine pancreatic insufficiency is a disease in which there is loss of pancreatic function. This can occur due to chronic pancreatitis, idiopathic atrophy of the pancreatic acinar cells, and very rarely, pancreatic duct obstruction or tumor formation. The damage to the pancreas causes loss of lipase, trypsin, and amylase production and decreases production of bicarbonate, which aids in absorption of Vitamin B 12; this leads to malabsorption of nutrients. Signs of pancreatic insufficiency include large-volume gray or yellow cow-patty-like diarrhea that occurs frequently throughout the day. The dog often produces gas, has episodes of diarrhea, loses weight, has a ravenous appetite, and may become

cachexic. Other signs may include unthrifty hair coat, pica, and coprophagia. This condition appears to be hereditary in German shepherds.

Exocrine pancreatic insufficiency is caused by loss of pancreatic function that leads to maldigestion and associated digestive disturbances. Diagnosis is based upon history, clinical signs, including frequent loose stools, diarrhea, weight loss, and muscle mass loss, and serum blood testing. Routine blood and chemistry testing usually are normal. If the animal does have high blood and urine glucose with signs of exocrine pancreatic insufficiency, concurrent diabetes mellitus should be considered. Some dogs have decreased levels of Vitamin B12 (cobalamin) and/or Vitamins A and E. To definitively diagnose exocrine pancreatic insufficiency, a serum sample should be taken after the animal has fasted to determine the concentration of trypsin-like immunoreactivity. Trypsin is considered to originate only in the pancreas and is an indicator of pancreatic problems; in cases of exocrine pancreatic insufficiency, trypsin concentration will be low.

Chronic Small Bowel Diarrhea

The differential diagnosis of a dog with chronic small bowel diarrhea consists of five broad categories: small intestinal disease, dietary issues, metabolic disturbances, infectious diseases of the gastrointestinal tract, and exocrine pancreatic insufficiency. Small intestinal disease can be caused by bacterial overgrowth secondary to loss of normal flora due to drugs or toxins or idiopathic causes. Motility disorders such as increased transit time can cause diarrhea. Tumors, inflammation, obstructions, congenital abnormalities, and lymphangiectasia of the small intestine can interfere with absorption and secretion. Dietary intolerances or food allergy can cause a dog to have diarrhea. Diseases that damage the liver or kidneys or hypoadrenocorticism can cause small intestinal diarrhea. Infections with parasites (Histoplasmosis or Giardia), bacteria (Salmonella), or mycobacteria can lead to diarrhea originating in the small intestine. Another cause of small bowel diarrhea in the dog is exocrine pancreatic insufficiency.

Aortic Stenosis

The dog can suffer from three types of aortic stenosis: valvular aortic stenosis, supravalvular aortic stenosis, and subaortic or subvalvular aortic stenosis. Of these three, the most commonly encountered type is subaortic stenosis. Regardless of type of stenosis, the obstruction causes increased stress on the ventricular wall. This affects left ventricular emptying, leading to left ventricular hypertrophy in an attempt to normalize ventricular systolic function. Hypertrophy then creates the potential for ischemic areas to form on the myocardium, resulting in mitral regurgitation and the development of

ventricular arrhythmias. Some dog breeds that are predisposed to aortic stenosis are large breed dogs such as the Newfoundland, the Boxer, the Rottweiler, the German shepherd, and the Golden Retriever.

Heartworm

Clinical signs in a dog with heartworm infection depend on worm burden, how long the animal has had the infection, how the immune system reacts to the presence of live and dead worms, and the amount and type of damage caused by the infection. Some dogs have no symptoms; other dogs develop signs such as exercise intolerance, coughing, difficulty breathing, epistaxis, lethargy, and ascites. The dog may develop increased lung sounds and heart murmur. Chest x-rays may reveal heart and pulmonary artery enlargement and lung densities. The animal may be anemic, have eosinophilia, basophilia, or hyperglobulinemia, and have protein spill over into the urine. Definitive testing is done via antigen detection; the antigen is generally detected six to seven months post-exposure.

Ehrlichiosis

Canine ehrlichiosis is caused by the rickettsia, *Ehrlichia canis* , which infects the monocytes and lymphocytes. The disease can either be acute or chronic and can cause a number of signs. It is spread by the brown dog tick *Rhipicephalus sanguineus* . Clinical signs include a recurrent fever, anemia, subcutaneous hemorrhages, bleeding from the nasal passages, thick nasal discharge, loss of weight, lymphadenopathy, swollen spleen, arthritis in multiple joints, central nervous system disturbances, and/or paralysis. The disease is diagnosed by clinical signs and laboratory testing by serum antibody testing and buffy coat and regular smears. The dog may also have neutropenia, non-regenerative anemia, thrombocytopenia, and hyperglobulinemia. Treatment is best accomplished through the administration of tetracycline, oxytetracycline, and doxycycline.

Herpesvirus 1 Infection

Canine herpesvirus 1 is a virus carried by many dogs but can cause a fatal sickness in neonatal puppies less than one month of age. The virus attacks the puppies either in utero or shortly after birth and damages the liver, kidney, lungs, and other body tissues. The clinical signs of infection in the puppy include green feces, refusal to feed, belly pain, difficulty breathing, and possibly death. In adult dogs, the clinical signs include mild nasal discharge and inflammation of the conjunctiva. Occasionally, there will be signs of a reproduction infection (discharge, blister-like lesions on the genitalia, and infertility). Diagnosis is based upon clinical signs and virus isolation/antibody staining of affected tissues. The only treatment is supportive care.

Influenza

An illness that is only recently emerging is canine influenza. It is caused by the influenza A virus (possibly from an equine strain) and is highly contagious; airborne or fomites spread the disease. Canine influenza was first discovered in Florida greyhounds in 2004 and has now spread to institutions such as boarding kennels, veterinary clinics, and animal shelters. Elderly dogs and puppies seem to be more susceptible to the disease, which causes signs such as coughing, fever, nasal discharge, loss of appetite, lethargy, and difficulty breathing. Most dogs present with a mild disease that runs its course in one to three weeks. Occasionally, pneumonia due to bacterial infection complicates the disease process. Treatment is primarily supportive care, although antibiotics should be used in cases of secondary bacterial infection.

Leptospirosis

Canine leptospirosis is caused by various *Leptospira* types: *interrogans* (serovars *icterohaemorrhagiae* and *canicola*), *grippotyphosa* , *pomona* , and *bratislava* . The disease has a wide range of signs, with some infected dogs appearing perfectly healthy and others having a fulminant acute disease course. The disease is transmitted by dogs carrying *Leptospira canicola* or by rodents (mice and rats) that carry *Leptospira icterohaemorrhagiae* . The acute disease is caused by the spirochetes attacking various body organs: the liver, kidney, spleen, and lymph nodes. The dog will start to vomit, refuse food, and have a fever. Liver and spleen damage may cause icterus and kidney damage renal failure, which is the most severe sign of infection. The gastrointestinal tract can be damaged as well, leading to continued vomiting and bloody stools. Treatment is good nursing care along with antibiotics, such as penicillin G, erythromycin, streptomycin, ampicillin, or chloramphenicol.

Lyme Disease

Lyme disease (or Borreliosis) is a disease caused by the spirochete *Borrelia burgdorferi* . It is spread by the *Ixodes dammini* tick, which becomes infected while feeding on infected small mammals. The organism causes a number of syndromes such as kidney problems, cardiac abnormalities, neurological disorders, and joint and limb disease. The most common clinical signs exhibited by the dog are fever, lameness, painful and swollen joints, loss of appetite, depression, and swollen lymph nodes. Renal disease is the second most common manifestation of Lyme disease and usually leads to death. Diagnosis is based upon clinical signs and laboratory testing. Treatment consists of a 10- to 14-day course of doxycycline or amoxicillin. Prevention includes adequate control of the tick vector and vaccination.

Review Video: Lyme Disease
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Nocardia Asteroides

Nocardiosis is a disease caused by a gram-positive, aerobic bacterium *Nocardia asteroides* . The organism is found in the soil, enters the body through wounds or by inhalation, and causes a chronic disease of cats and dogs along with other animals. Dogs are prone to develop a thoracopulmonary disease characterized by loss of appetite, weight loss, and fever. As the disease progresses, the animal may develop pleuritis or peritonitis along with abscess formation in the brain, heart, liver, and kidneys. Another form the infection takes is the mycetoma or skin form. After infection through a wound, a lump forms that will rupture and weep fluid. More areas of infection form and may eventually coalesce into draining sinuses. Along with these lesions, the animal may have a fever, be depressed, refuse to eat, and lose weight.

Cherry Eye

Cherry eye is caused by the prolapse of the gland associated with the third eyelid. In a normal dog, this gland is anchored to the periorbita beneath the third eyelid. Some breeds of dogs have a weak attachment, which predisposes them to a prolapse. Younger dogs are also usually afflicted with this condition. Breeds prone to developing cherry eye include the beagle, bulldog, bloodhound, cocker spaniel, Lhasa apso, and Shih Tzu. The condition can occur in just one eye or both eyes can be affected. The prolapse resembles an oval red mass protruding out from under the third eyelid. Irritation of the mass will lead to tearing, reddened conjunctiva, and eye spasms.

Gastric Dilation and Volvulus

Gastric dilation and volvulus is a syndrome that occurs in dogs that is frequently associated with large meal ingestion. The stomach fills with gas and twists around its axis, making it impossible for gas or food to exit the stomach. The stomach itself can be damaged due to local ischemia, compromised blood supply, or rupture of the stomach wall if pressure becomes too great. Other organs can be affected as well. The twisting of the stomach may squeeze the portal vein and caudal vena cava, obstructing blood flow from the abdomen to the heart. The expanding stomach can also press on the diaphragm, leading to breathing difficulty. These factors make gastric dilatation and volvulus syndrome an emergency that may lead to shock and cardiovascular failure.

The cause of gastric dilation and volvulus syndrome is not entirely understood. Some factors that predispose a dog to developing the syndrome include the

breed of the dog. Large deep-chested breeds are more susceptible to develop the syndrome, such as the Great Dane, Weimaraner, St. Bernard, Irish setter, Gordon setter, or Golden retriever. Other factors may include feeding one large meal a day, older age, intense activity and stress, or previous splenectomy. Signs that a dog may be suffering from gastric dilation and volvulus syndrome include unproductive retching, a distended abdomen, drooling, increased heart and breathing rates, anxious appearance, refusal to lie down, and signs of shock, such as weak pulse, pale mucous membranes, and slow capillary refill time.

Gastric dilation and volvulus syndrome is diagnosed based upon the clinical signs at presentation and radiographs. A right lateral abdominal radiograph of a dog with a gastric dilation and volvulus will reveal a distended gas-filled stomach that occupies most of the abdomen. The stomach may also appear as a double bubble, and may actually appear compartmentalized due to the twisting of the stomach around the central axis. Other testing is usually postponed during the acute phase, but blood testing will reveal an acute inflammation, along with electrolyte abnormalities and acid/basic disturbances. Other disease conditions that mimic gastric dilation and volvulus syndrome include abdominal foreign bodies, torsion of the spleen or mesentery, hernias, or abdominal neoplasia.

Mammary System Tumors

The female dog has five mammary glands running in two separate rows from the thorax to the inguinal region. These run from the thorax to the inguinal area, or cranial glands, and caudal thoracic, cranial, and caudal abdominal and inguinal mammary glands. More than half the tumors in female dogs are mammary tumors and about half of these are malignant. Tumors are more common in female dogs over eight years of age, and most tumors start in the two most caudal sets of mammary glands. If a female dog is spayed before her second estrus, it will reduce the chance of acquiring mammary tumors to less than ten percent; spaying before the first estrus will reduce the risk to .05 percent because mammary tumors are influenced by estrogen and progesterone.

Elbow Dysplasia

Elbow dysplasia is a general term that means elbow joint arthritis associated with developmental causes. There are four types of elbow dysplasia: ununited anconeal process, osteochondritis dissecans, fragmented coronoid process, and elbow incongruity. These diseases can occur separately or in any combination. Dysplasia is frequently common in both elbows; it is an inherited disease and is found in young dogs four to 18 months of age. Large and giant breed dogs are predisposed to developing elbow dysplasia. The breeds frequently represented

include Rottweiler, Newfoundland, German shepherd, Labrador retriever, Golden retrievers, Chow, bearded collie, and Bernese mountain dog. Other risk factors for developing elbow dysplasia include feeding a high calorie diet to dogs predisposed to the disorder or rapid weight and growth.

Osteochondritis dissecans (OCD) occurs when a piece of cartilage forms a flap or becomes fully detached from the bone; it affects the distal humerus at the medial trochlear ridge. An ununited anconeal process occurs when a bone fragment of the anconeus process fails to unite with the ulna (olecranon) during the first five months of life. A fragmented medial coronoid process occurs when a bone fractures or fissures off the ulna (the medial coronoid process). An elbow is considered to be incongruent when the elbow joint is malaligned and malformed. This predisposes the affected animal to abnormal cartilage wear and unnatural mechanical forces on the elbow joint. All these conditions lead to pain and arthritic changes in the elbow joint.

Hip Dysplasia

Canine hip dysplasia is a developmental disease of the hips initially manifesting as joint laxity in the young dog and progressing to malformation and degeneration of the coxofemoral joints, which may lead to degenerative joint disease (osteoarthritis) in the older dog. It is one of the most common skeletal disorders in the dog and tends to occur in large breed dogs, such as Labrador retrievers, German shepherds, Rottweilers, St. Bernards, and Golden retrievers. It has a strong genetic component and can also be influenced by other factors, including diet (energy and calcium levels), rapid weight gain, and by the pelvic muscle mass. The hip joint laxity can lead to subluxation of the femoral head and the acetabulum.

A dog with hip dysplasia has a history of difficulty rising from a sitting or prone position, has reduced its exercise, seems reluctant to climb stairs or jump, may display a “bunny-hopping” gait, and may have hind limb lameness that gets progressively worse. Physical examination reveals pain when the hip joint is manipulated, laxity of the hip joint, or “Ortolani sign,” which indicates joint laxity but is not diagnostic of hip dysplasia by itself), crepitus of the hip joint, and atrophy of the hind leg muscles. Radiographs (ventrodorsal with the hips extended) are used to assist in diagnosing hip dysplasia. Early hip dysplasia reveals a subluxation of the hip joint. As the disease progresses, the femur head flattens, the joint space narrows, bone spurs form on the femoral neck and joint capsule, subchondral sclerosis of the acetabulum and femoral head occurs.

Urinary Tract Infections

The dog's urinary tract is a common site of infection. Most infections involve the bladder, which normally holds sterile urine. A dog with a urinary tract infection may have the following signs: increased consumption of water, urinating frequently and in small amounts, inability to hold urine, and occasionally, bloody urine. Most cases of cystitis are caused by bacteria ascending up the urethra and into the bladder. It is uncommon for male neutered dogs to have a urinary tract infection caused by an ascending infection. To diagnose a urinary tract infection, a urinalysis test strip can be used or the urine can be cultured to discover what bacterial organism is causing the infection. The most common bacterial cause of urinary tract infection in the dog is *E. coli* .

Seizures

It is important to investigate the cause of seizures. Animals can have seizures either due to an event occurring outside the brain or because of a primary brain disorder. Conditions outside the brain that can cause seizures include poisons, hypoglycemia, toxins, or medical conditions. Primary brain dysfunction can be caused by infections, trauma, or neoplasia. It is important to rule out these conditions as the cause of seizures. If none of these conditions exist, then the animal may have epilepsy. A common cause of seizures for animals less than one year of age is a brain infection. Animals older than five years of age commonly have tumors that cause the seizures. Epilepsy with no known cause is most likely to be diagnosed in dogs between one and five years of age. Certain breeds of dogs, such as Collies, Cocker Spaniels, Basset Hounds, and Schnauzers, are more prone to developing epilepsy.

Review Video: Seizures
Visit mometrix.com/academy and enter code: 977061

Hypothyroidism

Dogs can be affected by hypothyroidism, which is usually the result of destruction of the thyroid gland. The two conditions that destroy the thyroid gland in the dog are idiopathic atrophy of the thyroid gland and lymphocytic thyroiditis. Lymphocytic thyroiditis is an autoimmune disorder in which the thyroid gland is infiltrated by macrophages, lymphocytes, and plasma cells that destroy the follicles and cause the gland to become fibrotic. Dogs over the age of four years and spayed females are more likely to have the disease. Early clinical signs include weight gain, exercise intolerance, dry skin, shedding, lethargy, and thinning hair. As the condition worsens, the skin may thicken due to deposition of glycosaminoglycans in the dermis; this is most pronounced in the face giving the animal a "tragic" expression. Treatment is with replacement thyroxine (T4) at the dose of .01 to .02 mg/lb daily, divided into two doses.

Birds and Poultry

Respiratory Tract

The respiratory system of the bird begins with the nasal openings, which open into the nasal cavity. The nasal cavities are connected to the sinuses, which are around each eye. The nasal cavities serve to filter air before it reaches the lungs. A slit at the roof of the mouth allows air to pass between the nasal cavities and the lower respiratory system. The larynx is at the rear of the mouth and connects the trachea to the gullet. At the lower end of the trachea, the larynx divides into two bronchi that connect with the lungs. Both the trachea and bronchi are composed of cartilage rings. The lungs of birds are small, red, sponge-like structures. Air sacs surrounding the internal organs of the bird increase lung capacity and serve as air reserves; these thin membrane-like structures are often the first area attacked by respiratory disease.

Digestive Tract

From the mouth, the digestive tract travels into the body via the esophagus. At the lower end of the esophagus is the crop, which functions as temporary storage for food. The last portion of the esophagus connects to the stomach. The stomach is composed of two parts: the proventriculus and the gizzard. The gizzard connects to the small intestine. The pancreas lies along the first part of the small intestine. At the lower end of the small intestine, two ceca are attached. The cecal bacteria function to help break down food. After the ceca, the small intestine passes into the large intestine, which empties into the cloaca, a common collecting point of digestive, reproductive, and urinary products. The cloaca opens to the outside of the body at the vent. Accessory organs to the digestive tract are the liver and the gall bladder; these structures are connected to the small intestine through the bile duct.

Reproductive, Urinary, and Internal Organs

The urinary, reproductive, and digestive systems all empty into a common collecting area called the cloaca. The reproductive system in the female bird includes the ovary and the oviduct. The ovary consists of a collection of egg yolks of varying sizes that lies near the kidneys. The oviduct coils down from the ovary to the cloaca. In the male, the testes are oval organs lying between the lungs and kidneys. Ductus deferens runs from the testes to the cloaca. The urinary system in the bird begins with the two kidneys; each kidney has a ureter, or tube, which passes waste products from the kidneys to the cloaca. The heart and spleen are the vascular organs of the bird. The heart has four chambers and the spleen is a roundish dark red organ lying between the liver and gizzard.

Feather Picking

Feather picking is a serious disease in which the bird plucks, chews, or amputates its own feathers. A bird with this disorder is prone to infections and has trouble regulating its body temperature. Because the body responds to feather picking by trying to create new feathers, the bird will have increased nutritional demand, possibly resulting in debilitation, suppressed immune system, lethargy, and a decreased life span. Some owners react to the denuded bird negatively as well. Feather picking can be caused by medical disorders, such as metabolic/organic disease, parasites, neoplasia, infections (viral, fungal, or bacterial), pain, or malnutrition. A medical condition may lead to behavioral feather picking, but behavioral feather picking may also be caused by other stimuli such as a lack of attention, stress, changes in environment or household, hormones, or sexual frustration.

A bird with a suspected case of feather picking needs to be examined to be sure that it does not have a medical condition. One sign that the bird has a feather picking problem is that the feathers on the head are spared because the bird cannot reach these feathers. Diet changes or nutritional deficiencies can have a deleterious effect on a bird's overall health. The environment (such as the cage, toys, bedding, fumes, tobacco smoke, and other birds/pets in the house) can negatively affect the behavioral attitude of a bird prone to feather picking. Most pet birds are fairly social animals; paying less attention to the bird (or even if the bird just perceives less attention), changes in household members, and past breeding behavior can all lead to stress and feather picking.

Pacheco's Disease

Pacheco's disease occurs in birds and is caused by a number of closely related herpes viruses. The virus infects B or T lymphocytes, nerve cells, and epithelial cells. It is highly infectious and is transmitted by the fecal-oral route. The virus passes out of the body of an infected bird in feces and dried nasal discharges. The virus remains viable in the contaminated dust, which becomes aerosolized. Symptoms of Pacheco's disease include depression, ruffled feathers, sinusitis, diarrhea, anorexia, tremors, and conjunctivitis. The feces may become green with urates, indicating that the liver has been damaged. Diagnosis is performed by polymerase chain reaction, histopathology, and sequence testing for the virus DNA. Birds that survive the disease should be considered carriers.

Sinusitis

Birds are commonly affected by sinusitis. Amazon and African gray parrots are prone to develop this disease. Diseases or pollutants that block the sinus passages stop the normal flow of mucus, which usually clears pathogens from the sinuses, leading to sinus infection. Signs of sinusitis include nasal and eye

discharge, sneezing, facial swelling, and unkempt appearance. Some nutritional deficiencies have been implicated in the development of sinusitis, such as vitamin A, vitamin C, and zinc deficiency. Parasites, fungus, viruses, and bacteria can also cause sinusitis in the pet bird. In addition, low humidity levels can predispose a bird to develop sinusitis.

Parasites

Caged pet birds suffer from many types of parasites. Among these are protozoal diseases such as coccidia, toxoplasmosis, and cryptosporidiosis, as well as nematodes and cestodes. Another parasite affecting pet birds is *Trichomonas gallinae* , characterized by mucosal lesions of the mouth that appear as white to yellow material adhering to the mucosa. Birds of prey can also be infected with *T. gallinae* , which is spread through direct and indirect contact. Giardia, a protozoan, is spread among birds by eating the infective cysts. The infected birds have bubbly, large-volume stools; it has been suggested that some birds exhibit feather picking under the wings and the thigh in response to an infection.

Avian Influenza

Avian influenza (AI) virus has emerged as a significant concern, as it is a zoonotic disease that infects birds. There are two forms of AI: low pathogenic (LP) and high pathogenic (HP). Signs of LP AI include swollen sinuses and eye and nasal discharge; LP AI also infects the lower respiratory tract. Birds appear sick and layers stop laying eggs. It is a fairly mild disease and does not cause high death rates unless aggravated by other infections or poor husbandry. HP AI causes significant mortality; some birds may die suddenly, while other show pre-death signs such as blue and swollen combs and head, bloody nasal discharge, and diarrhea. If a bird does survive the initial infection, it will frequently develop secondary CNS signs such as ataxia, torticollis, or opisthotonos.

The avian influenza virus is a type A orthomyxovirus. The virus is further divided into the subtypes 15-hemagglutinin (H1-15) and 9-neuraminidase (N1-9) and occurs in either a low pathogenic (LP) or a high pathogenic (HP) form. Most of the viruses are of the LP type, while a few of the H5 and H7 subtypes are mutated and fall into the HP category; these subtypes affect domestic poultry. After an incubation period varying from three to seven days, birds transmit the virus to other birds that ingest or inhale the virus. The virus can also be carried on fomites such as shoes and clothing. The LP viruses are fairly common and can be found throughout the world, although most commercial flocks are free of the virus. The mutated HP form, however, causes devastating losses in certain areas of the world.

Histomonas Meleagridis

Histomonas meleagridis is a protozoon that is transmitted by *Heterakis gallinarum* , a nematode that inhabits the ceca of birds. The disease has been called blackhead. Unlike other birds that may be infected but do not die, turkeys usually die from the infection. The life cycle of *H. meleagridis* begins with the larva of earthworms infected with *H* . *gallinarum* that harbor *H. meleagridis* . Birds eat the worms or eggs infected with *H. gallinarum* and become infected with *H. meleagridis* ; *H. meleagridis* is then released and multiplies in the ceca, burrowing into the tissues and causing damage. The histomonads travel to the liver either through the circulatory system or via the peritoneal cavity and damage the liver tissue and may also combine with other digestive pathogens. Clinical signs of infection include yellow stool, depression, ruffled feathers, and drooped wings.

Common Worm Species

There are many worm species that infect birds; the most significant are the nematodes, or roundworms. The most common roundworm is *Ascaridia galli* , a small intestinal worm found in chicken, ducks, turkeys, and quail. The eggs of *A. galli* pass out of the chicken in its stool. Provided good conditions exist, the eggs will become infected in 10 to 12 days. A bird ingests the eggs, which hatch into larva in the proventriculus; these larva travel to the duodenum and live in the lumen for about nine days. After this time period, they burrow into the mucosa, return to the lumen in 17 days, and mature in the lumen at approximately 28 days. The larva in the tissue cause significant bleeding and damage.

Newcastle Disease

The virus that causes Newcastle disease is avian paramyxovirus-1, an RNA virus. Newcastle disease is primarily a respiratory disease. Clinical signs in an infected bird or flock occur within 12 days after exposure to the aerosolized virus. It can be also spread via the fecal-oral route. Respiratory signs include cough, increased lung sounds, sneezing, and open mouth breathing. There can also be digestive signs (diarrhea) or signs attributed to the nervous system. A form of Newcastle disease called neurotropic velogenic disease combines both respiratory and nervous system signs. Another form, called viscerotropic velogenic Newcastle disease (or exotic Newcastle disease), is characterized by respiratory signs, swelling of the head and neck, depression, anorexia, and diarrhea.

Dissecting Aneurysms

Young, rapidly growing turkeys (primarily males from two to six months of age) may be afflicted with dissecting aneurysms that cause sudden death. The aneurysms can develop anywhere in the vascular system and can burst, causing

fatal internal bleeding. The cause is not known but is suspected to be a combination of genetics and diet. A diet that encourages rapid growth, such as a high-fat diet, may cause plaques to form in the blood vessels. High blood pressure on these plaques may cause a fatal rupture. Apparently healthy birds are usually found dead; necropsy reveals an anemic carcass and large quantities of clotted blood in the body cavity. A frequent site of rupture is the posterior aorta. While the exact cause of dissecting aneurysm is not known, increasing dietary copper levels (125 to 250 ppm) and limiting feed intake at four to six months of age may help lessen the incidence.

Performing a Necropsy

Wash the dead bird with detergent water. Place the bird on a flat surface with the breast up and the head extended away from you. Remove the upper portion of the beak by cutting through the nasal cavities and turbinate bones with shears. Check for signs of infection, squeezing to extrude exudates. Examine the eyes for signs of inflammation. Cut through one corner of the mouth and extend the cut down the neck to examine the area for abnormalities or disease. Cut the larynx and trachea and open to examine for abnormalities or signs of disease. Next, begin examination of the abdomen by nicking the skin right below the tip of the breast bone. Make a cut around the body to the sides. Peel the skin over the breast to expose muscles and examine the muscles for abnormalities. Cut the abdominal skin where the legs join the body. Dislocate the femoral joints by pressing on each leg, and then peel the skin from the legs for examination.

Being careful not to cut too deeply, make an incision into the abdominal muscle immediately beneath the breast bone. Extend the cut to the back towards the wing attachments. Push the breast up and dislocate the shoulder joints, cut through the joints, and remove the breast; examine the air sacs. Examine the internal organs before removing the liver, heart, and spleen; make incisions in the tissues to examine internal surfaces. Examine the exposed intestinal tract, and then sever near the gullet and behind the ceca to remove. Cut the crop to examine tissue, then wash and examine the internal surface. Cut the proventriculus and gizzard for examination; slit the intestines lengthwise and examine the tissue and lining. Examine the reproductive organs, kidneys, and ureters, and then remove from the body. Examine the lungs and bronchi, then the sciatic and brachial nerves.

Diagnostic Tools

General Chemistry Panel

A chemistry panel should include the following measurements of various blood components in order to identify the most common clinical conditions

affecting the large animal: Albumin, Albumin/globulin ratio, Aspartate aminotransferase (AST; GOT), Glucose, Blood urea nitrogen (BUN), Creatinine, Creatine phosphokinase (CPK), Sorbitol dehydrogenase (SDH), Gamma-glutamyl transferase (GGT), Bilirubin (to include direct, indirect, total), Total protein, Total carbon dioxide, Sodium, Potassium, Calcium, Phosphate, Venous blood gases

The horse has a higher bilirubin concentration than other large animal species. BUN is a less reliable indicator of renal function in ruminants because the microflora in the rumen metabolizes urea nitrogen. Horses and cattle both have lower GGT values than burros and donkeys.

Reasons for Variation

There are a number of reasons why normal laboratory values vary when testing the large animal. These include factors associated with using a particular laboratory, variations between the different species, differences within a particular species, sex of the animal being tested, and age of the animal being tested. Test results need to be interpreted by taking into account all of these factors. It is generally preferred to use a veterinary diagnostic laboratory over a human medical facility in order to obtain the most accurate results. A laboratory will return test results with a normal reference range for their particular tests. It is extremely important when submitting a laboratory sample to carefully note the age, sex, species, and breed of the animal being tested in order to obtain accurate readings and appropriate reference ranges.

Liver and Renal Panel

In the large animal, blood chemistry values that measure liver and biliary function and damage should be included in the liver panel, such as albumin, aspartate aminotransferase (AST/GOT), albumin/globulin ratio (A/G ratio), alkaline phosphatase (AP), bilirubin (direct, indirect, total), blood glucose, blood urea nitrogen (BUN), fibrinogen, gamma-glutamyl transferase (GGT), sorbitol dehydrogenase (SDH), and total protein. Blood chemistry measurements to check for renal disease or dysfunction include BUN, calcium, creatinine, and phosphate. For both liver and renal testing, it is also recommended that urinalysis be performed to measure specific gravity, bilirubin, casts, cells (including RBCs and WBCs), occult blood, and protein.

Serum Enzyme Elevations

The following are common causes of serum enzyme elevations in the large animal:

Sorbitol Dehydrogenase (SDH): Acute liver failure, chronic liver failure, liver abscesses, damage to the bowel

Gamma-Glutamyl Transferase (GGT): Acute and chronic liver failure, liver flukes, cholangiohepatitis, cholelithiasis, aflatoxicosis, pyrrolizidine alkaloid toxicity

Lactase Dehydrogenase (LDH): Muscle disease (down cow, malignant hyperthermia, malignant edema), degenerative nutritional diseases, rhabdomyolysis (tying-up, azoturia, myositis), acute and chronic liver failure, cholangiohepatitis, hemolysis, cholelithiasis

Aspartate Aminotransferase (AST/GOT): Muscle disease, acute and chronic liver disease, liver flukes, cholangiohepatitis, cholelithiasis

Creatine Phosphokinase (CPK): Malignant hyperthermia, malignant edema, down cow, degenerative nutritional diseases, exertional rhabdomyolysis (tying-up, myositis, azoturia)

Alkaline Phosphatase (AP): Acute and chronic liver disease, cholangiohepatitis, cholelithiasis, liver flukes, pyrrolizidine alkaloid toxicity (**Note** : not a good test for ruminants.)

Staining

Simple stains consist of aniline dyes derived from coal tar. They fall into two groups: basic and acid stains. The basic simple stains are crystal violet, basic fuchsin, gentian violet, methyl violet, methylene blue, and safranin. Acid stains are used to stain the cytoplasm of bacteria; examples include acid fuchsin, brilliant green, and Congo red. **Gram staining** consists of staining the sample with gentian violet or methyl violet, treating the slide with Gram's or Lugol's iodine solution, and then decolorizing the slide with a solvent (acetone or alcohol). The slide is then washed and counter-stained with neutral red, safranin, or Ziehl's fuchsin. Those bacteria that retain the violet color are Gram-positive; those that take the counterstain are Gram-negative.

Gram-Negative Facultative Anaerobic Rods

Gram-negative facultative anaerobic rods are important causes of digestive tract diseases and other disease conditions and consist of two families: the Enterobacteriaceae, which become mobile using peritrichous flagella, and the Vibrionaceae, which become mobile using polar flagella. The Enterobacteriaceae family includes the genera *Citrobacter, Edwardsielleae, Enterobacter, Ernimia, Escherichia, Hafnia, Klebsiella, Proteus, Salmonella, Serratia, Shigella* , and *Yersinia* . The Vibrionaceae family includes the *Vibrio, Aeromonas, Plesiomonas, Photobacterium* , and the *Lucibacterium* genera. Bacteria can be further classified according to the number and arrangement of flagella, very delicate structures that give the bacteria motility. These bacteria

may be divided into four main groups: monotrichous, which have a single flagellum, lophotrichous, which have two or more flagella, amphitrichous, which have a single flagellum at each end, and peritrichous, which have flagella arranged around the entire cell.

Tissue Samples for Food Animals

In cattle that have died of respiratory disease, lung tissue from both abnormal and unaffected lobes should be submitted to the laboratory in formalin and as fresh tissue. The liver, spleen, kidney, and heart should be submitted as fresh tissue samples. The formalin-preserved tissues can be checked for IBR, BVD, and BRSV by immunohistochemistry. The fresh tissues can be PCR tested for BVDV, IBRV, PI3V, BRSV, and Mycoplasma. In the pig, tissues that should be submitted in formalin include those of the lung (diseased and normal tissue), liver, spleen, kidney, and heart. Fresh tissue should be submitted from diseased and normal lung. In addition, a swab of the nasal turbinates should be submitted for culture, along with a cross-section of the snout if atrophic rhinitis is suspected.

Urinalysis

Urine samples are collected for urinalysis either midstream, by manual expression, through catheters, or by cysto centesis. Normal 24-hour urine production in both the cat and dog is 20 to 40 mL per kg body weight. Polyuria is an increase in urine production, oliguria is a decrease in urine production, and anuria is absence of urine production. Normal urine appears in various shades of yellow; other colors can occur such as red tint due to blood in the urine or green tint due to bilirubinuria. Urine is clear in healthy animals, except for the horse, which has thick, cloudy urine. In small animals, cloudy urine indicates the presence of casts, cells, or crystals. Urine will usually have a characteristic odor, but strong- or foul-smelling urine suggests bacterial infection; a sweet smell can indicate ketosis or presence of glucose in the urine.

Specific gravity ranges from 1.001-1.060 in the normal animal. If the kidneys cannot concentrate the urine, the specific gravity will be near the glomerular filtrate value (1.010). The term isosthenuria refers to urine that has not been concentrated in the tubules and has the same specific gravity as plasma; The term hyposthenuria refers to urine that is dilute. Normal pH is between six and eight in animals and depends upon diet. Herbivores have more alkaline urine, whereas carnivores have more acidic urine. In a normal animal, there should not be any glucose, ketones, or blood in the urine. Bilirubin may be present in small amounts in the urine of healthy dogs but should not be present in cat urine. A small amount of protein may be present in concentrated urine. Usually,

there is a small amount of sediment in the urine; sediment can be red or white or occur as epithelial cells, bacteria, casts, or crystals.

White Blood Cell Values

When interpreting white blood cell values, a number of factors must be considered. If there is evidence of inflammation (eosinophilia, monocytosis, and/or an increased number of immature neutrophils, termed a left shift), the total white blood cell count may be low, normal, or high. High neutrophil counts (greater than 25,000/µL) also indicate an inflammatory response by the body. Stress can also cause a slight decrease in lymphocytes (750/µL to 1500/µL) and eosinophils, as well as minor elevation of neutrophils and monocytes. The normal neutrophil to lymphocyte ratios in domestic species are .5 to 1 in ruminants, .7 to 1 in pigs, 1.1 to 1 in horses, and 3.5 to 1 in cats and dogs.

Radiographs

A radiograph is an image made by the interaction of photons with the silver on the radiograph film to produce a latent image. When the film cassette is treated with developing fluids, this latent image becomes a black and white image. The white is termed opacity and is determined by the density and thickness of the object being radiographed. Bones, soft tissues, air, fat, and metal are all opaque. An artifact is an image that is not real but is produced by problems with the silver granules on the film or by blurring. A radiograph is considered to be a diagnostic legal document. As such, it should be carefully labeled with positioning, the side on which the animal was placed, patient name, the date, and name of the clinic.

Thoracic

Radiographs of the thorax will give the clinician useful information on a number of conditions that affect the thoracic cavity, including lung disease, cancerous growths affecting the thorax, and heart disease. Animals that exhibit the following clinical signs can benefit from radiological examination of the thorax: coughing, wheezing, vomiting, respiratory distress, heart murmurs, regurgitation of food, or cyanosis. Radiographs are useful in emergency medicine as well as in cases of trauma or wounds to the chest. Thorax radiographs will also give information regarding the chest wall, neck, pleura, and diaphragm. The structures typically seen in a thoracic radiograph in a small animal include the lung, cervical and thoracic spine, heart, blood vessels, mediastinum, ribs, diaphragm, forelimb, trachea, esophagus, stomach, liver, sternum, and fat and soft tissue.

Conventional radiology is an anatomic tool used to detect and diagnose disease in the abdomen, pelvis, chest, and bones. Fluoroscopy involves the use of a

continuous beam of x-ray to evaluate dynamic processes such as the movement of the gastrointestinal tract or may be used in interventional procedures such as catheter placement. Tomography uses an x-ray tube and film that move in opposite directions while the film is exposed; this produces a sharp image in different planes of the body. Radiographs can be combined with contrast to produce a sharper contrast in images that may not be clearly seen on plain x-ray. Common contrast agents are barium, iodides, or water-based agents.

Ultrasounds

Ultrasound is a non-invasive diagnostic tool that uses high-frequency sound waves greater than 20 kHz. When passed over the body, a transducer emits and receives the sound waves; gel is used on the body to displace air that would reflect the ultrasound beam. The sound waves emitted by the transducer are reflected by body tissues and travel back to the transducer; the transducer then converts the sound waves into an electrical signal and becomes amplified. Each amplitude is assigned a shade of gray; stronger waves are white while weak ones are black. A special type of ultrasound known as Doppler is used to evaluate vascular flow; this technique uses the Doppler effect to detect frequency shifts in the reflected beam.

Health Maintenance and Problem Management

General Conditions and Diseases

Escherichia Coli

E. coli is an important disease-causing organism in animals; it causes diarrhea and septicemia in young animals and edema disease in pigs. *E. coli* has also been found in abscesses and in cases of pneumonia and can cause urinary tract disease. The cell is adapted with special properties that make it better able to infect or damage the host, such as special hemolysins and siderophores that disrupt the host cell. Adhesins (pilli or fimbria) are special proteins which make the bacteria attach to other cells; different strains of *E. coli* have their own adhesins. The capsule lends extra protection to the cell membrane against the body's immune system. Enterotoxins take control of the host's nucleotide activity, which regulates water and electrolyte secretion by the cell.

Transudates

A transudate is normal fluid that accumulates in excessive amounts. A transudate may have a low total protein count (less than three grams per deciliter) and a low nucleated cell count of less than 500 cells per μL; however, the horse can have a normal nucleated cell count of up to 10,000. The most common cause of a transudate is venous statis, which is associated with

diseases such as liver failure, kidney disease, cancer, and congestive heart failure. A modified transudate is one in which dead mesothelial cells are found in the effusion and attract further cells; this increases the total protein and nucleated cell count to 3.0 to 5.0 and 5000, respectively. Exudates have even higher protein and cell counts (3.0 to 7.0 and up to 100,000, respectively) and are usually caused by inflammation, which in turn causes vascular damage.

Nematodes

Nematodes, or roundworms, are significant animal parasites that exist in many different species. Structurally, nematodes are very simple organisms; adult nematodes have approximately 1000 somatic cells. Nematodes possess nervous, digestive, reproductive, and excretory systems but do not have respiratory or circulatory systems. The digestive system consists of an alimentary canal that runs from the mouth to the anus. Although nematodes exist in separate sexes that are morphologically different, the size and shape of the species vary greatly. Nematodes range in length from .3 mm to well over 8 meters; their life cycle can either be direct (host to host transmission of larva or eggs) or indirect, requiring intermediate hosts. Depending on species, the eggs are quite hardy and are resistant to temperature and disinfectants.

Cestodes

As adults, tapeworms, or cestodes, inhabit the intestinal tracts of vertebrates. Generally, the life cycle includes an intermediate host such as earthworms, snails, insects, or crustaceans, but the definitive host is always a vertebrate. The body of a tapeworm has an anterior scolex that the cestode uses to attach to the host. The rest of the body is composed of a long posterior strobila divided into many segments called proglottids. The primary organ system of the tapeworm is the reproductive system; the rest of the organs are very small or absent. There is no digestive tract; instead, the tapeworm absorbs nutrients across the body surface via the syncytial neodermis, which is composed of many microvilli to enhance absorption. The length of the tapeworm ranges from less than 4 mm to well over 12 inches; tapeworms are hermaphroditic and may fertilize by self- or cross-fertilization.

Veterinary Epidemiology

The following are some terms used in veterinary epidemiology to describe disease occurrence. An **endemic occurrence** is a predictable disease state. It is used to describe the constant presence of a disease within a given population and the usual frequency at which a disease occurs in a given population. It does not imply that a disease is expressing clinical signs in the population. **Epidemic occurrence** is an epidemic of disease at a level in excess of the endemic level. A

pandemic is a widespread epidemic that hits a substantial part of a population. A **sporadic disease occurrence** is one that is irregular and happens at a local level; an outbreak is a disease occurrence.

Antibodies

Antibodies are produced by the lymphocytes of the body. They act as a primary defense against the invasion of the body by foreign substances. Antibody production is stimulated when an antigen enters the body. There are five classes of antibodies: **M, G, E, A, and D** . These are combined with the term immunoglobulin (Ig), which is another name for an antibody. IgM is the first antibody produced by neonates and the first one produced when the animal has an infection. IgG is produced when the body is exposed to the same antigen for a second time and is the predominate antibody in the body. IgE is produced in response to allergens. IgA is found in the saliva and in mother's milk. The function of IgD has not been defined.

Lymphocytes

The lymphocyte is a type of white blood cell consisting of B cells and T cells. The B cell produces antibodies, an important component of the immune system in neutralizing foreign substances. T cells attack bacteria, toxins, and viruses and help to regulate other parts of the body's immune system. Lymphocytes are the agents involved in cellular immunity. They are made in the bone marrow and multiply in the spleen and thymus. Lymphocytes circulate in the vascular system and penetrate the walls of the vascular system to reach the tissue cells. They return to the bloodstream via the lymphatic system to the jugular and subclavian veins. Lymph nodes in the lymphatic system collect and manufacture lymphocytes.

Cytokines

Cytokines are low-molecular weight proteins that have local, potent effects in the immune response to tissue damage and inflammation. They modulate cytotoxic and antigen-specific responses through paracrine communication (between cells that are in close proximity to each other) and endocrine communication. Only a small quantity of cytokines is produced by the cells. Cytokines act on the target cell by triggering the specific receptors on the target cell surface. There are many different cytokines that are now classified into groups, including the interleukins (1 to 15), interferons (alpha, beta, and gamma), colony-stimulating factors, fibroblast growth factors, tumor necrosis factors, and transforming growth factors. Cytokines have also been implicated in certain disease conditions such as cancer, sepsis, and arthritis.

Passive Transfer of Immunity

Passive transfer of immunity is needed in animals because although they have an immune system, they may not have the antibodies they need when they first encounter pathogens after birth. The mother passes antibodies to the offspring via transfer of passive immunity. This is done either in the uterus by passing the antibodies through the placenta or following birth through the colostrum. Colostrum is a thick, sticky yellowish fluid that is rich in immunoglobulins. The major immunoglobulin in colostrum is IgG. When a young animal ingests colostrum, the proteins reach the small intestine and bind to the intestinal epithelial cell receptors. The IgG is taken up by the cells and is then taken to the bloodstream, passing on the mother's immunity to her young.

Nutrition

Necessary Nutrients

Nutrients are substances that when ingested support life. Carbohydrates are starches, sugars, and fiber. Mammals produce enzymes to digest starches and sugars. Ruminants can also digest fibers due to microbial action in the rumen. Fatty acids give feed or food flavor and provide high levels of energy. Amino acids are the basic elements of protein. There are 22 amino acids, which are needed for protein synthesis; 12 can be made by the body, but 10 are considered essential amino acids and must be ingested. These essential amino acids are arginine, histidine, isoleucine, lysine, methionine, phenyl aniline, threonine, tryptophan, and valine. Glycine is an essential amino acid for chickens; taurine is essential for cats. Water is a vitally important nutrient in all animals. Minerals, including calcium, chlorine, magnesium, phosphorus, potassium, and sulfur, and microminerals, including copper, iodine, iron, manganese, selenium, and iron, are important in many physiological processes. Biochemical reactions are catalyzed by vitamins.

Fat-Soluble Vitamins

Vitamins are essential nutrients needed by the body in small amounts and are divided into two groups: fat-soluble (A, D, E, and K) and water-soluble (B-complex and C). The fat-soluble vitamins are stored in the body in the liver and fatty tissues. Because of this storage, there is a greater risk of toxicity when they are given in excessive amounts. Vitamin A is needed for the formation and health of skin, mucous membranes, night vision, and bone and tooth development. Vitamin D is needed for bone and teeth hardening and helps to increase the absorption of calcium. Vitamin E is an antioxidant and helps to prevent damage to the cell membrane. Vitamin K is essential in blood clotting.

Water-Soluble Vitamins

Vitamins are divided into two groups: the fat-soluble vitamins (A, D, E, and K and the water-soluble vitamins B-complex and C, and are essential nutrients in body functions. The water-soluble vitamins are not stored by the body and need to be replenished on a daily basis. The B-complex vitamins are thiamin (B1), riboflavin (B2), niacin (B6), Vitamin B12, folate, biotin, and pantothenic acid and serve as co-enzymes in many physiological functions. Vitamin C (ascorbic acid) has a number of important functions in the body, including wound healing, strengthening the walls of blood vessels, improving iron absorption and utilization, and immune system functioning.

NSAIDs

Nonsteroidal anti-inflammatory drugs (NSAIDs) are used to treat acute and chronic pain and inflammation, to reduce fever, to relieve perioperative pain, and to treat cardiovascular disease. They can be used alone or combined with other drugs such as narcotics to give the patient a synergistic analgesia. The mode of action of most NSAIDs is to inhibit cyclooxygenase enzymes that catalyze the incorporation of oxygen into arachidonic acid. This stops the production of cyclooxygenase-1 (COX-1) and cyclooxygenase-2 (COX-2), mediators of inflammation. COX-1 is expressed in all body tissues and is used in the formation of prostaglandins; COX-2 is found in inflamed and damaged tissues and intensifies the inflammatory response. Therefore, NSAIDs work as anti-inflammatory and pain-relief agents by inhibiting COX-2. This inhibition of both cyclooxygenase enzymes can impair platelet production and stop the protective effect of prostaglandins, leading to digestive tract ulceration.

The most commonly administered NSAIDs and their dosages are as follows:

Acetaminophen: For use in dogs only: 10 to 15 mg per kg orally every six to eight hours.

Aspirin: Dogs: 10 mg/kg orally every 12 hours.

Aspirin: Cats: 10 mg/kg orally every two to three days.

Carprofen: Two to four mg/kg (PO, SQ, IM, IV) every 12 to 24 hours.

Deracoxib: Dogs only: three to four mg/kg orally every 24 hours.

Etodolac: Dogs only: 10 to 15 mg/kg every 24 hours.

Ketoprofen: Dogs: .5 to 1 mg/kg every 12 hours; cats: same dose every two to three days (PO, IM, SQ, IV).

Ketorolac: Dogs only: .25 to .5 mg/kg every 12 hours (IM, IV, SQ).

Meloxicam: Dogs: .1 to .2 mg/kg orally every 24 hours; cats: same dose every two to three days.

Piroxicam: .3 mg/kg orally every 48 hours.

Review Video: NSAID Effects
Visit mometrix.com/academy and enter code: 569064

Anticonvulsant Agents

Anticonvulsant agents used in veterinary medicine include phenobarbital, bromide, and diazepam. These agents are commonly used to suppress seizure activity. Phenobarbital is used as a maintenance drug in dogs. Some adverse effects of phenobarbital include sedation, liver damage, increased drinking, eating and urination, and physical dependence on the drug. Bromide (either potassium or sodium salt) is used with phenobarbital or in animals that cannot tolerate phenobarbital. Some adverse side effects include bronchial asthma in cats, nausea, sedation, stomach upset, increased drinking, eating, and urination, and neurological signs if too much of the drug are given. Diazepam is used to control status epilepticus in dogs or used with phenobarbital or bromide. It is the primary choice to control seizures in cats and can be used in foals as well.

Penicillin

Penicillin antibiotics include penicillin G and V, amoxicillin, ampicillin, carbenicillin, cloxacillin, hetacillin, nafcillin, and ticarcillin. The penicillin antibiotics have bactericidal effects on susceptible gram-positive and gram-negative organisms. The bactericidal action is via disruption of cell wall synthesis. The drug is widely distributed throughout the body and the bones with the exception of the brain and CNS (if they are not inflamed). The drug can cause hypersensitivity reactions, such as rash, fever, anaphylaxis, hives, and joint pains; sensitivity to one type of penicillin means the animal is sensitive to all types. Amoxicillin is commonly combined with clavulanate potassium (Clavamox) to protect the amoxicillin from β-lactamase-producing staphylococci. Mixing penicillin with aminoglycosides causes both drugs to become inactivated. Use with tetracyclines, chloramphenicol, and erythromycin antagonizes the bactericidal activity of penicillin.

Aminoglycoside Antibiotics

Aminoglycoside antibiotics are used to treat gram-positive, gram-negative, and mycobacterial infections. They are especially effective against aerobic gram-negative bacteria, including *Enterobacter* , *Escherichia coli* , *Proteus* , and

Klebsiella . Among the aminoglycoside antibiotics are gentamicin, amikacin, dihydrostreptomycin, neomycin, kanamycin, tobramycin, and streptomycin. Some of the aminoglycoside antibiotics are also useful against *Pseudomonas* infections. They work by interference with the reading of the mRNA transcript of the bacteria, resulting in the production of nonsense proteins. Because they are highly polar and have difficulty crossing biological membranes, they are poorly absorbed from the digestive tract. Ototoxicity, nephrotoxicity, and neuromuscular blockade (weakness and respiratory arrest) are the most commonly encountered adverse side effects from use of aminoglycoside antibiotics.

Lincosamides

Lincosamides include the antibiotics lincomycin and clindamycin. Lincomycin is active against gram-positive cocci and penicillinase-producing staphylococci. Clindamycin is also active against gram-positive cocci and often against other gram-positive organisms resistant to penicillin and the cephalosporins. These drugs are widely distributed throughout the body and bones and cross into the placenta of some species but do not readily cross into the cerebrospinal fluid (CSF). Both are metabolized in the liver, clindamycin more so than lincomycin. Some adverse affects include vomiting and diarrhea, pain after injection of the drug, and potentiation of the effects of neuromuscular blocking agents. In some cases, horses, rabbits, and humans have suffered fatal diarrhea after the use of lincomycin. It advisable not to use lincosamides in the horse because of the potential to develop fatal colitis and it should not be used in neonates because of the limited capability of the neonate to metabolize pharmaceutical agents.

ACE Inhibitors

Angiotensin-converting enzyme (ACE) inhibitors inhibit the formation of angiotensin II. This is produced when the kidneys release renin, an enzyme that acts on angiotensinogen to produce angiotensin I. Angiotensin I is then acted upon by angiotensin-converting enzyme to form angiotensin II. Angiotensin II stimulates the release of aldosterone from the adrenal cortex, which subsequently causes the body to retain sodium and water; it also causes vasoconstriction. ACE inhibitors, such as enalapril, captopril, lisinopril, and benazepril, are balanced vasodilators, which prevent vasoconstriction, reduce preload and afterload, and reduce sodium and water retention. They are useful in cases of congestive heart failure and systemic hypertension. Some adverse side effects of the ACE inhibitors include hypotension, especially when used with diuretics or other vasodilators, as well as kidney problems, vomiting, anorexia, or diarrhea.

Beta-Adrenergic Drugs

Beta-adrenergic drugs are used to attenuate the effect of the sympathetic activity on the myocardium. The sympathetic nervous system affects the heart by increasing conduction velocity, heart rate, rate of relaxation, oxygen demand, and excitability. Catecholamine is released, which can cause arrhythmias on compromised heart muscle. Beta-blockers block the actions of the catecholamines epinephrine and norepinephrine on the beta-adrenergic receptors. Some types of beta-blockers include atenolol, propranolol, sotalol, and esmolol. These agents are commonly used in practice to treat cardiac arrhythmias and hypertension and to protect the heart after a heart attack. Other uses of beta-blockers include treatment of mitral valve prolapse, congestive heart failure, hypertrophic obstructive cardiomyopathy, glaucoma, and atrial fibrillation.

Anesthesia

Acepromazine

Acepromazine, a commonly used phenothiazine, is a potent neuroleptic agent and acts as a depressant on the central nervous system. It is quick-acting and is used as a sedating agent that also has muscle-relaxing properties. It is frequently used in dogs, horses, and cats. In small animals, acepromazine is used to sedate fractious animals, as an anti-vomiting medication for car sickness, and as a preanesthetic agent. It is used in horses to tranquilize fractious animals and is combined with local anesthetics for surgical procedures. In male horses (especially active stallions), it induces paralysis of the retractor penis muscle that causes persistent paraphimosis or priapism. The common dosage is as follows: dogs, .25 to .5 mg/lb; cats, .5 to 1 mg/lb; horses, 2 to 4 mg/100 lb.

Atropine and Glycopyrrolate

Atropine is a competitive antagonist of muscarinic cholinergic receptors and is used as a premedication for anesthesia. Along with its use as a premedication, it is also used to treat sinus bradycardia, incomplete atrioventricular block, and sinus block or arrest. As a parasympatholytic agent, it causes digestive tract, biliary tract and urinary tract relaxation, and suppresses secretions from the gastric, respiratory, and salivary systems. It is used to decrease ocular inflammation as well as a mydriatic and cycloplegic agent. Another use for atropine is in the treatment of organophosphate and carbamate intoxication. **Glycopyrrolate** is another anticholinergic medication with twice the potency of atropine but does not increase the heart rate as much as atropine; it has the

added benefit of not crossing the blood-brain barrier, and its effects last longer than those of atropine.

Thiobarbiturates

Thiobarbiturates are very short-acting barbiturate anesthetics used to induce anesthesia or for very short surgical procedures. The reason they are ultra short-acting drugs is that once introduced into the bloodstream, they are almost immediately distributed to various tissues and finally to the fatty tissues. Deposition into the fat depots along with slow metabolism of the drug result in a prolonged recovery time following administration, although return to consciousness is quick. Some side effects of thiobarbiturate use include depression of respiratory centers (apnea), inadvertent subcutaneous or perivascular injection leading to skin sloughing or irritation, cardiac arrhythmias, and a decrease in intracranial and intraocular pressure. In horses that are not premedicated with a phenothiazine prior to use of a thiobarbiturate, recovery from anesthesia can cause violent excitement.

Ketamine

Ketamine is a nonbarbiturate, dissociative anesthetic used for chemical restraint or anesthesia of short duration. It has a quick onset of action, allows the animal to maintain normal muscle tone and laryngeal reflexes, increases intraocular and intracranial pressures, provides analgesia, stimulates the cardiovascular system, and causes some respiratory depression. Some adverse effects of ketamine are as follows. An injection can be given in the muscle, but it will cause pain. In procedures requiring muscle relaxation, ketamine should be combined with another anesthetic agent, as it increases muscle tone. It should not be used in animals prone to seizures, as the drug has been known to cause seizures. Because this drug is excreted by the kidneys, caution should be used in animals with known kidney disease.

Xylazine and Medetomidine

Two commonly used α2-agonists in veterinary medicine are xylazine and medetomidine; both have potent analgesic power. These drugs can cause sedation and general anesthetic states. They produce their effects by aggravating α-adrenergic receptors. Xylazine has a rapid onset of action and provides good short-term analgesia and sedation, and recovery after administration is smooth. The effects of medetomidine are longer-lasting than those of ketamine. Some adverse effects of these two drugs include cardiovascular depression, bradycardia, atrioventricular heart block, vasoconstriction, increased intrauterine pressure, increased urine output, hyperglycemia, and vomiting. These drugs have the benefit of being reversible.

Ketamine and medetomidine can be reversed by the use of atipamezole (Antesedan) or yohimbine.

Anesthesiology Terms

Analgesia: Relief from pain.

Anesthesia: Relating to the process of producing insensibility. Provided by anesthetic agents or drugs that produce a loss of consciousness and a lack of motor response to irritating stimuli.

Basal anesthesia: Produced by preanesthesia agents in preparation for a deeper plane of anesthesia by other anesthetic agents.

General anesthesia: Produces a level of unconsciousness by controlled yet reversible intoxication of the central nervous system.

Hypnotic: A narcotic that induces a state of unconsciousness from which an animal can easily be aroused.

Local anesthesia: Use of a substance to cause a loss of sensation to a localized area of the body.

Narcotic: Agents that depress central nervous system activity.

Sedative: A narcotic substance that calms a subject and causes drowsiness.

Surgical anesthesia: A level of anesthesia that causes unconsciousness and muscle relaxation.

Tranquilizer: Produces sedation without drowsiness.

Fractures

Fractures can have problems healing due to three types of conditions: a delayed union, a nonunion, or a malunion. Delayed union is considered to happen after a certain amount of time has passed in which it would be reasonable to assume the bone should have healed. The causes of a delayed union include inadequate reduction of a fracture, infection, loss of blood supply, inadequate immobilization, or distraction. A delayed union can lead to a nonunion. A nonunion occurs when the bone has stopped healing without a bony union. A malunion occurs when the bones heal in an abnormal position; these are further classified as functional malunion in which the bone maintains normal function and nonfunctional malunions, which incapacitate patients.

Pins

There are three types of pins used in veterinary practice: the Steinmann pin, the Rush pin and the Küntscher nail. The Steinmann pin is the most commonly used. The Steinmann pin performs best when used for short oblique or transverse fractures in the middle third of the long bones. The Steinmann pin is often used with cerclage wiring to increase the stability at the fracture site. In more complicated fractures, multiple Steinmann pins are used. The Rush pin is used in pairs to repair fractures of the distal humerus and distal femur and to repair the central third of the humerus or femur diaphysis. The Küntscher nailing technique is used to repair short oblique, transverse, or middle third fractures of the humerus, tibia, and femur as long as no longitudinal cracks or bony cortex fissures are present.

Screws

Screws are used in conjunction with plates to provide a reliable form of internal fixation. They can be used singly for femoral head fractures or for distal humerus and femur intercondylar fractures. The two basic types of screws are the fully threaded cortical screw and a partially threaded cancellous screw. The cortical screw provides interfragmentary compression and is used in most fracture fixation cases. They should not be used for some fractures in the young, growing animal, in those with metabolic bone disease, or in those with osteoporosis. The cancellous screw works to compress interfragmentary fractures by having the threads on one side of the fracture exert the force against the head of the screw resting on the other side of the fracture.

Fractures on Radiograph

Radiographs of the bone should include at minimum two views. The exposed film should include the joints above and below the fracture and if there is soft tissue swelling, the exposure should be increased. Signs seen on the radiograph that indicate a fracture include the disruption of the normal bone contour, radiolucent fracture lines, free bone fragments, foreign material, and an increase in opacity due to fragment or overlapping fracture ends. Fractures are classified as closed or compound, complete or incomplete, chip or slab, avulsion, transverse, spiral, oblique, irregular, articular or non-articular, stress, fatigue, impaction, or compression or luxation. A unique type of fracture is the Salter-Harris fracture, which is a fracture of the unfused growth plates. Neutered male cats can have these fractures at a more advanced age, as their growth plates do not fuse as soon as other animals. These fractures can lead to abnormal healing.

Slings and Dressings

Stabilizing Joints and Fractures

An Ehmer sling is used when stability is needed after a reduction of a dorsal-cranial dislocation of the hip joint. This sling provides abduction and internally rotates the femur and flexes the knee. The Velpeau sling is used on the shoulder to keep the carpus, elbow, and shoulder joints in a flexed position and to prevent weight-bearing of the forelimb. It is used to keep weight off the forelimb, to immobilize scapula fractures, and after shoulder joint dislocations. A Schroeder-Thomas splint is used to immobilize fractures that occur distal to the midhumerus and midfemur. This is a traction-type splint used to put the fracture in proper position and to counteract muscle forces while aligning and immobilizing the fracture.

Robert Jones Dressing

A Robert Jones dressing is a temporary bandage used to immobilize fractures especially if there is severe swelling or edema associated with the swelling; it is also used after knee surgeries. A stirrup is applied to the dorsal and ventral foot surfaces, and then one-pound rolls of cotton are firmly wrapped around the limb to form a bulky bandage. Approximately two rolls are required for a 50-pound dog. The bandage is further compressed through the use of a roll of conforming bandage (vet wrap) or elastic tape. Over the course of the next 10 days, the bandage loosens and must be changed after this time period if it is still needed. The Robert Jones dressing must be kept dry to prevent skin maceration.

Luxation of the Patella

Patellar luxation occurs when the patella, which normally rides in the femoral groove within the stifle, "pops" out to the inner aspect of the knee joint. The patella is a small bone situated in the tendon of the quadriceps muscle (the extensor muscle.) The patella inside the tendon and the quadriceps work to extend the leg. In animals with a luxating patella, the patella slips out of alignment when the leg is flexed. While the condition can occur in any breed of dog, the congenital condition is more common in the small breeds. About half the animals have the condition in one knee, while the other half have it in both knees. In addition to congenital causes, luxation is occasionally caused by trauma.

Patellar luxation can be classified in order to direct treatment for dogs suffering from this condition. In Grade 1, the patella can be luxated (medially or laterally) manually. The patella then returns to its normal position in the trochlear groove when it is released. In Grade 2 luxations, manual manipulation of the patella or flexation of the stifle causes the patella to luxate. The patella remains luxated until it is manually replaced. Grade 3 animals have a patella that remains luxated, but can be reduced manually; however, it will return to the luxated

position once manual pressure is released. Grade 4 patellar luxations remain luxated at all times and manual reduction is impossible.

The cause of patellar luxation is not entirely known unless the knee has suffered trauma. In nontraumatic cases, dogs have a shallow trochlear groove where the patella normally rides. Deformities associated with this condition include bowing of the limbs, an increased laxity of the stifle joint, rupture of the cranial ligament, and internal rotation of the tibia. Other disorders that may be associated with patellar luxation include: hip dysplasia, cranial cruciate problems, abnormal conformation, especially of the femur and tibia, an excessively long patellar ligament, or a very tight quadriceps muscle. Besides luxation, other signs associated with patellar luxation include lameness and pain. Many animals with Grade 1 and Grade 2 patellar luxation may only have occasional pain. Those with Grade 3 and Grade 4 patellar luxation may have constant pain, lameness, and physical deformities.

Antiseptics

Common antiseptics used to prepare animals for surgery include alcohols, chlorhexidine, chlorinated phenols, diphenyl ether, halogen-substituted phenol, iodophors, and quaternary ammonium compounds. Alcohols denature proteins and prohibit mitosis; they work very rapidly and are effective against bacteria, fungi, viruses, and mycobacterial. Chlorhexidine works by increasing cell wall permeability and is effective against bacteria, fungi, enveloped viruses, and yeasts. Chlorinated phenols inhibit membrane enzymes and disrupt the cell wall of gram-positive bacteria. Diphenyl ether inhibits fatty acid synthesis and disrupts the cell walls of bacteria. Halogen-substituted phenols are effective against bacteria, enveloped viruses, and some fungi; these work by inactivating enzymes and disrupting cell walls. Iodophors inhibit protein synthesis of bacteria, mycobacterial, fungi, yeast, protozoa, and yeast. Quaternary ammonium compounds are primarily effective against gram-positive bacteria by denaturing proteins and impairing permeability.

Sutures

Suture material is chosen to provide a secure wound closure while minimizing reaction to the foreign material placed in the body. Synthetic sutures have a lower incidence of infection in contaminated wounds. These include monofilament nylon, polyglactin, polyglycolic acid, and polypropylene. Absorbable sutures can either be natural, such as catgut or collagen, or synthetic, such as polyglycolic acid, polyglactin 910, polydioxanone, polyglyconate, or poliglecaprone 25. Nonabsorbable sutures include natural materials such as cotton and silk, stainless steel, or the nonabsorbable synthetic

sutures, such as nylon, polymerized Caprolactam, polybutester, polyester fibers, polypropylene, or polyethylene. Absorbable sutures generally lose their strength and degrade within 60 days of placement. Nonabsorbable sutures retain their strength for over 60 days.

Adherent Dressings

Adherent dressings can either be a dry-to-dry type of dressing or a wet-to-dry dressing. The dry-to-dry dressing is wide-meshed gauze applied to wounds having foreign material, a low viscosity exudate, or loose necrotic tissue. The fluid passes through the mesh into a secondary absorbent layer while the debris and necrotic tissue stick to the gauze to allow removal of these substances when the bandage is removed. A wet-to-wet dressing is used in wounds with numerous loose debris and a high viscosity exudate. Wide-meshed gauze is first soaked in sterile solution and then placed over the wound; this is covered with a secondary absorbent layer and a third layer. The wound exudate is then absorbed by the first and second layer. This bandage is changed when the first layer dries, usually in 24 hours, taking with it the loose debris and necrotic tissue.

Wound Drains

Drains are used to draw gas or fluids away from a wound and to eliminate dead space. Drains can complicate wound healing if caution is not used. The can become channels for ascending infections into the wound and can cause incision dehiscence if they are not properly placed. Drains can either be passive or active devices. A passive drain relies on gravity and pressure to take fluids or gas away from the wounds. Examples of these are red rubber, polypropylene, and Silastic or soft latex (Penrose) drains. Active drains are those that have a negative pressure applied through use of suction. These can be used if more fluid needs to be drawn away from the wound, if there is a large amount of dead space to close, or if bandage fluid saturation needs to be reduced.

Gunshot Wounds

Gunshot wounds that remain superficial are frequently encountered in practice and are particularly prevalent in rural areas. These wounds are classified as contaminated wounds and make good environments for bacterial growth, as many times extensive tissue and blood supply damage occur. The tissue damage is classified as laceration and crushing injury. The projectile can also cause shock wave damage and cavitation injury. These two forces cause concussion, which causes further vascular injury and tissue damage and can drive foreign material deeper into the body. Gunshot wounds to the skin and immediate underlying tissue should be generously debrided, lavaged, and bandaged and

the animal should be started on broad-spectrum antibiotics. More extensive wounds need to be surgically debrided.

Wound Healing

Wound healing can be complicated by infection, dehiscence of the incision site, and the formation of seromas or hematomas. Bacteria can be introduced into the wound at time of wounding, during surgical procedures, or after wound closure. Bacterial infection occurs predominantly in wounds with a compromised blood supply, in animals with poor health, in the presence of large amounts of necrotic tissue, blood, or foreign material or because of poor surgical technique. Wounds that become infected need to be debrided, lavaged, and left open to heal by second-intention or closed at a later date. Antibiotic therapy is mandatory in these cases. Fluid can collect between tissue layers in wounds with dead space or in wounds with seeping of blood. The seromas or hematomas can be reduced by use of drains and careful attention to hemostasis during wound closure.

Salivary Mucoceles

Salivary mucoceles can be drained if they are causing life-threatening breathing problems; however, drainage will not cure the condition and surgery is warranted to stop the saliva from collecting in the mucocele. Most mucoceles originate from the sublingual glands or ducts. Therefore, surgery usually involves removal of these glands. Typically, the submandibular salivary gland is removed as well because it is in close proximity to the sublingual glands. If the mucocele is caused by the zygomatic gland, this gland should be removed. Complete removal of the glands contributing to the mucocele resolves the problem. Complications include swallowing difficulties, recurrence, or infection. Recurrence indicates that the resection was incomplete or that the wrong gland was resected.

Nerve Blocks

Metacarpal and Metatarsal Nerve Blocks

Metacarpal and metatarsal nerve blocks are useful procedures in order to perform lower foot surgery on the bovine. The nerves blocked during the metacarpal block are the superficial radial nerve branch, the dorsal ulnar nerve branch, and the medial and palmar nerves. The nerves blocked during the metatarsal nerve blocks are superficial and deep peroneal nerves and the medial and lateral plantar metatarsal nerves. At the anatomical sites corresponding to these nerves, 5 mL of 2% lidocaine is injected. The animal should be restrained properly and the area for infusion of the anesthetic agent should be thoroughly cleansed. If necessary, hair should be clipped to remove

caked-on debris or excessive hair. The needles used to block the nerves should be new and sharp. A light dose of sedation should be used; however, this may make determining the effect of the nerve block more difficult.

Medial Digital Nerve Block

A useful nerve block technique to learn in the bovine is the medial digital nerve block. This block will successfully dull sensation for removal of corns or interdigital vegetative dermatitis. The animal should be given preoperative sedation and be placed in lateral recumbency and restrained before attempting to inject the anesthetic agents. The limb to be injected should also be properly restrained, the entire claw and pastern should be surgically scrubbed, and antiseptic should be applied. A two- to three-inch 18-gauge needle should be applied to a 20-cc syringe and 20 mL of 2% lidocaine with epinephrine should be drawn. Remove the needle and plunge it full length midline into the interdigital tissue immediately proximal to the pastern joint on the dorsal surface. Infuse 10 cc of lidocaine in this area; then remove the needle and repeat on the plantar surface.

Peterson Nerve Block

A Peterson nerve block is used to enucleate eyeballs and blocks the nerves C-III, C-IV, and C-VI, the maxillary and ophthalmic branch of C-V, and the auriculopalpebral branch of nerve C-VII. A 4.5-inch 18-gauge needle, a 20-cc syringe, and 2% lidocaine are used to perform the block. The needle is inserted into the depression posterior to the point where the supraorbital process meets the zygomatic arch until the tip of the needle strikes the coronoid process. Sweep the needle up along the process until it hits the bony plate of the pterygopalatine fossa floor. Retract the needle about ¼ of an inch and draw back on the plunger to make certain no blood is present in the hub. If no blood is present, inject the syringe contents. Retract the needle to just under the skin and infuse 3 cc of lidocaine just above the zygomatic arch.

Sacral Paravertebral Nerve Block

A sacral paravertebral block is used in cattle, sheep, goats, and pigs. It is used for such procedures as replacing a rectal or vaginal prolapse, to counteract the straining associated with enteritis, proctitis, or cystitis, and to treat vaginal urine pooling. In cattle, sheep, and goats, the goal is to block the S4 nerve. This is commonly done by using alcohol solutions. In pigs, this type of nerve block should be used for both the S3 and S4 nerves. Lidocaine is used in the pig to block the nerve. In all species, a successful nerve block is indicated by lack of straining of the animal, full desensitization of the perineal area, and normal tail function.

Ich

Ich is a common disease of freshwater fish caused by the encysting protozoa *Ichthyophthirius multifillis* . Scale fish are particularly prone to developing this problem. It is the feeding stage (trophozoite) that feeds on the skin or gills of fish. After it has fed, the trophozoite falls off the fish and forms a tomont, or an encapsulated dividing stage. The capsule adheres to objects in the aquarium environment and divides. Water temperature plays an important role in this disease. It generally has an outbreak in water temperatures between 59 to 77 °F (15 to 25 °C). The protozoan forms small white spots over the fish skin. The parasite destroys the fish skin when it feeds, making it susceptible to secondary infections. Treatments that are commonly used are chloramines and quinine.

Filtration Systems for Fish

Filtration systems for fish can be biological, mechanical, chemical, ultraviolet, or other types. Biological filters work by using bacteria to break down the solid and liquid wastes; they also break ammonia and nitrites down into nitrates. The underground gravel filter uses this type of filtration system. Mechanical filters uses filter wool or foam to remove the solid wastes from the water. Ultraviolet filtration is used to kill potential pathogens and to keep them from spreading from system to system. Chemical filtration systems use material such as activated charcoal to remove dissolved wastes from the water. Other types of filters include the rapid sand filter, protein skimmers, ozone, and the trickle filter.

Measuring Disease Frequency

Measuring disease frequency is based upon the prevalence and incidence of disease. Prevalence is the frequency of existing cases in a population that is occurring at any given point in time. Incidence is defined as the occurrence of new cases that arise in a given time period in a specified population. Measuring prevalence and incidence requires counting the cases in the population at risk (i.e., the number of cases). Sometimes the term “attack rate” is used interchangeably with incidence while a disease outbreak is occurring. It is calculated by number of people affected by the disease with the number of people who were exposed to the disease. Prevalence and incidence are usually converted to rates (number of cases divided by the population number.)

Controlling Seizure Activity

The use of medication to suppress seizures is weighed against the potential toxic effects the medication can have on the animal. A rule of thumb to follow before beginning anti-seizure medication in an animal is to wait for seizures to

become more frequent than once a month or to begin medication if seizures occur in clusters. The most commonly prescribed drug to control seizures in dogs is Phenobarbital; this is a barbiturate that must be given for two weeks before the drug reaches levels capable of controlling seizures. Biannual blood levels should be tested to determine a phenobarbital blood level. Because this drug may adversely affect the liver, liver function should be tested twice yearly. Another drug used to control seizures in dogs is potassium bromide; this drug takes longer to become effective and can be given along with phenobarbital.

Toxoplasma Gondii Stages

Toxoplasma gondii is obligate intracellular coccidia that can infect mammals including humans; it causes the disease Toxoplasmosis. The cat is the definitive host; all other animals susceptible to infection are intermediate hosts. The organism has three infectious stages: Oocysts, which are shed in the feces, contain sporozoites. Tachyzoites are found in tissues and comprise the actively multiplying stage. Bradyzoites are also found in tissue in cysts and are a slowly dividing stage. *T. gondii* is spread by ingestion of tissues carrying the cysts or ingestion of water or food that has become contaminated with the oocysts; a fetus can become infected from an infected mother.

Classifications of Seizure Disorders

Epilepsy can be categorized according to the type of seizure disorder. Grand mal seizures (generalized) are the most common form in the small animal. The entire body is involved, with stiffness or a cycle of tonic/clonic action (stiffness and contraction cycling). During a grand mal seizure, the animal loses consciousness and may urinate and/or defecate. Partial seizures occur in one specific area of the body. Partial seizures can become generalized seizures as the condition progresses. Psychomotor seizures are characterized by abnormal behavior. The animal may howl, snap, have a frenetic activity burst, or circle. This behavior may then deteriorate into a generalized seizure. After the seizure, the animal appears disorientated. This is an important sign, as it can differentiate seizure activity from fainting; fainting is not followed by disorientation

Classification of Wounds

When presented with a wound, the surgeon should classify the wound as contaminated or uncontaminated. If it is contaminated, the degree of contamination must be determined. A clean wound is defined as a nontraumatic and noninflamed operative wound in which the gastrointestinal, genitourinary, respiratory, or oropharyngeal tracts have not been entered. A clean contaminated wound is an operative wound in which there is controlled

entry into the gastrointestinal, genitourinary, or respiratory tract or when there is a clean procedure performed in which a drain is placed. Operations on traumatic wounds and operations in which digestive tract contents or infected urine is spilled or in which there is a breakdown of aseptic technique are considered to be contaminated. A dirty wound is a wound in which operations are performed on necrotic tissue, foreign bodies, or purulent discharge or in which there is fecal contamination.

Skin Tumors

When a veterinary surgeon removes a skin tumor, the success or failure depends upon whether an adequate amount of the margin surrounding the tumor has been removed. It is not always known from simple observation if the tumor is completely excised. Normal-appearing tissue may have infiltrates of tumor tissue. The surgeon relies upon known tumor behavior to estimate how much of the normal tissue beyond the tumor should be removed. There are three general classifications of tumor margins: local excision involves removing the tumor and the tissue immediately adjacent to the tumor, a wide local excision involves removing the tumor along with a large margin of surrounding tissue, and radical excision involves removing the tumor, surrounding tissue, and tissue lying adjacent, such as blood vessels, lymphatics, muscle, and nerves.

Elbow Dysplasia

Elbow dysplasia, a term encompassing the four diseases of the joint, including the ununited anconeal process, elbow incongruity, the fragmented medial coronoid process, and osteochondritis dissecans, is usually treated with surgery. Diagnosis is based upon radiographs of the affected areas using different views, including the craniocaudal, mediolateral, mediolateral with the elbow hyperflexed, and oblique view of the elbow. With the ununited anconeal process, surgery is performed to either remove the loose bone fragment or fix the loose fragment to the ulna with a screw. Osteochondritis dissecans and fragmented coronoid process require removal of the loose fragments. Often this can be accomplished using arthroscopic surgery techniques. Elbow incongruity repair with surgery is controversial and depends upon degree of joint degeneration and surgical skill of the surgeon.

Glaucoma

Treatment of an animal with glaucoma begins with discussion with the owner that there is no cure for the disease. The medications used to treat glaucoma work by lowering the intraocular pressure. They are chosen based upon their effectiveness on the type of glaucoma from which the animal suffers. Medications include prostaglandin analogs, miotics, beta blockers, systemic

hyperosmotic agents, and carbonanhydrase inhibitors. Open-angle glaucoma is treated with miotics, carbonic anhydrase inhibitors, beta blockers, prostaglandin analogs, and osmotics. Some animals need both medication and surgery. Surgical options include intravitreal gentamicin/steroid injection, cyclocryothermy, shunt placement, and intrascleral prosthesis. Animals that have lost vision and are in pain may need to have the eye enucleated.

Cataracts

Cataracts are typically treated by removal of the lens. A common treatment is pharmacoemulsification with IOL implantation. It is usually an outpatient procedure performed under general anesthesia with a neuromuscular blockade. Prior to surgery, the eyes are treated with topical corticosteroids and antibiotics along with systemic nonsteroidal anti-inflammatory agents. An incision is made in the cornea and the anterior chamber is entered. The lens is then removed and an IOL is implanted. The incision is closed, topical medication is applied, and the animal is fitted with an Elizabethan collar. Topical medication is continued for three to four weeks postoperatively. Complications after cataract surgery are rare as long as postoperative instructions are followed.

Nocardiosis

Diagnosis of Nocardiosis is based upon the clinical signs (thoracopulmonary disease or skin lesions) along with demonstration of the organism in samples collected from the affected animal. Differential diagnoses include blastomycosis, cryptococcosis, and Actinomycosis, which can have similar skin lesions. The thoracopulmonary form can resemble other lung diseases. The organism should also be cultured using blood or Sabouraud's agar to give positive identification to the organism. Treatment of Nocardiosis is a long-term process, greater than three months, in order to eliminate the bacteria from the body. Deep-seated or widespread skin lesions may need to be surgically debrided and any deep infection may need to be drained. Antibiotics should be chosen based upon susceptibility testing. Some antibiotics that have been found to be effective against the organism are trimethoprim-sulfadiazine, amikacin, erythromycin, tetracyclines, sulfadiazine, and minocyline.

Bufotoxin Ingestion

The giant tropical toad *Bufo marinus* produce toxic substances called bufotoxins (conjugated bufagins) in their parotid glands. Dogs, and rarely, cats, ingest the toxin when they attempt to mouth the creature. The toxin, bufagin, has a cardiac effect similar to digitalis and causes ventricular fibrillation. Other toxins have pressor effects similar to catecholamines and hallucinogenic effects. The

affected animal has increased salivation and shakes its head. It may act blind, become ataxic, vomit, and have diarrhea. The respiratory rate will increase and the animal may experience convulsions. Immediately after mouthing the toad, the owner should rinse out the animal's mouth to remove toxins. The animal should then be treated with activated charcoal and a cathartic. Anesthetics should be administered as needed, taking into account the animal's overall health status.

Buckwheat Ingestion

Herbivores can be poisoned by buckwheat (*Fagopyrum esculentum*) within 24 hours after ingesting the plant. It is rarely lethal, but clinical signs can endure for days to weeks. Ingestion causes photosensitization of unpigmented skin; all parts of the plant are capable of causing this reaction. The actual toxin is not known but it is assumed to be caused by a fluorescent substance called fagopyrin; thus, the term fagopyrism is the name associated with the photosensitivity. Signs include inflammation and swelling around the face, itching, skin sloughing, vesicle formation, which may rupture and ooze fluid, erythema, and nervous signs. The ruptured vesicles may crust over, and in some cases, patches of necrosis will form on the skin.

Johne's Disease

Control and prevention of Johne's disease takes time and dedication. Because there is no cure for the disease, preventing the introduction of a Johne's-positive animal into a herd should be of paramount importance. Replacements to the herd should only be purchased from certified disease-free herds and semen used for artificial insemination should be from Johne's-free studs. Infected herds need to undergo an extensive test and cull program. Any adults with chronic diarrhea should be isolated, tested for Johne's, and culled if positive. All adults should have fecal cultures at half-year intervals. Positive animals should be culled. Strict hygiene should be followed to break the fecal-oral route of infection. Neonates should be separated from cows at birth and given colostrum from dams negative for the disease; newborns from Johne's-positive dams should be culled from the herd.

Generally, the tests available for detecting Johne's disease have a high specificity, or a low rate of false-positive results. The sensitivity of the tests is approximately 50%. 50% of infected animals test positive for Johne's; this is due to the fact that early in the disease course, animals do not test positive due to low shedding of the bacterium or a low immune response. There are eight tests available to detect Johne's disease: fecal culture, complement fixation test, agar gel immunodiffusion test, enzyme-linked immunosorbent assay test,

BACTEC culture, DNA probe, interferon, and a skin test. Four of the tests are blood tests for serum antibody, three of the tests detect *Mycobacterium paratuberculosis* , the causative agent of Johne's disease, and two tests are assays for cellular immunity.

Rabies

Rabies is caused by a Lyssavirus that produces encephalomyelitis in animals infected with the virus. The disease is characterized by one of two forms: a furious form or a dumb (paralytic) form. The Lyssavirus can have different variants that can be identified by monoclonal antibody analysis or through genetic sequencing. These variants cause rabies in various species, but they can all be transmitted to susceptible animals. In North America, variants are maintained in skunks, raccoon, wild canines, and bats. There is no known feline variant. Bats have been the most frequent cause of human rabies cases in the past few decades; in particular, the variant associated with the silver-haired bat and the Eastern Pipstrelle.

The rabies virus (Lyssavirus) is spread almost exclusively through the saliva of an infected animal. Most cases involve bites or scratches from a rabid animal. It is invariably fatal once clinical signs are seen in an animal infected by the virus. The virus can be killed by sunlight and common disinfectants but in the tissues, it can remain infective for weeks at normal or refrigerator temperatures. An animal can shed the virus for several days before development of clinical signs. The incubation period is very variable, but usually the animal develops clinical signs two weeks to three months after been exposed. After being inoculated by the virus, the virus travels up the peripheral nerves to the spinal cord and then to the brain. After it reaches the brain, the brain then sheds the virus, which then travels via peripheral nerves to the salivary glands.

Professional Behavior , Communication and Practice Management

Workers' Compensation

Workers' compensation is needed to offset losses to employees resulting from work-related injuries and also to hasten the injured worker's return to work. All staff members on the veterinary clinics payroll are covered by workers' compensation regulations and laws. Most states require employers to pay for the workers' compensation wage loss and medical benefits. As part of workers' compensation, employers are required to carry adequate insurance coverage and also to plan, prevent, and mitigate worker injuries. Veterinary clinic employees are exposed to many potential sources of injuries, such as animal

bites, chemicals, medications, scratches, kicks, and zoonotic disease. Focusing on mitigating these potential causes of injury and illness will help maintain employee health and lower workers' compensation claims.

Assistants and Receptionists

Veterinary assistants have less training than a veterinary technician or technologist. The AVMA has no approved credentialing process for veterinary assistants. While they may perform various duties at the veterinary clinic, many assistants are directed to feed and exercise the animals, clean and maintain kennels, and restrain and handle animals. They should receive training on proper animal care, handling, and any other duties to which they are assigned (radiographs, pharmacy) to decrease practice owner liability. Receptionists should be chosen for their position based upon their communication skills and ability to interact with clients. Receptionists should be trained in regard to office protocols and procedures to ensure agreement between what they relay to clients and what the veterinarians and technicians actually discuss with clients. They are important in the process of client bonding to the clinic and its staff.

Client-Based Practice

Veterinary medicine is considered to be a service industry. The service provided to clients is considered to be long-term service (the veterinary-client relationship). Since clients are not able to judge a veterinarian or the staff on the quality of their medical care, they generally judge a veterinary clinic on the quality of service it provides. A positive climate fostered in the veterinary practice spills over into the quality of service it provides. Clients want consistent, quality service at each visit. They want to feel special, but they also have different needs and desires at different visits. One important concept is that clients want all veterinary practice staff to care about their pets. This means remembering their pet's gender and name, showing affection, empathizing with the pet during uncomfortable procedures, and remarking on the pet's unique qualities.

Cost Estimates

When an animal presents with a medical or surgical condition that will likely incur large expenses, the clinic should routinely produce a cost estimate for the client. This will ensure that the client comprehends what work will be performed and how much the services provided will cost. By receiving an estimate, the client will then be able to discuss payment terms, ask questions about the services being provided, and discuss the need for recommended services. When discussing an estimate with a client, the clinic staff member

should ascertain if the client understands what is being said because the client may be under emotional stress at the time. Give the client time to ask questions and discuss concerns and treatment options. Have the client sign the estimate after discussion.

Employee Performance Review

Employee performance reviews are needed to let team members know where their strengths and weaknesses lie in regard to their work duties. The goal of a performance review is to implement changes that will lead to improved employee performance. The review process should be scheduled during an employee's regular work hours and should be done in private. A review should praise the employee's strengths, but also should include a two-way discussion about an employee's weakness and areas in which the employee's performance could be strengthened. It is also helpful to recognize a particularly positive behavior exhibited by the employee. The employee should be given a written evaluation and initialed copies should be kept in their personnel files.

Return on Investment

Return on investment is a measure used to evaluate the ability of a business to use assets to generate additional value for the people who own the business. In veterinary practice, it is a difficult value to ascertain and is not regularly used in practice valuation. Return on investment is calculated by dividing net income by the monetary value of the company. It is expressed as a percentage and the higher the percentage the more efficient the company is at using its capital to generate a profit. On tax returns, certain adjustments are taken to properly calculate the true profits of the company. These include compensation for management and medical/surgical work of the owner (s), depreciation of equipment, rent, and leases for equipment.

Radiation Safety

Radiation exposure in the veterinary clinic must be taken seriously by all those involved in taking and developing radiographs. Long-term exposure to even low doses of radiation has been linked to many disorders, including genetic, cancerous, glandular, and other disorders. High-dose radiation causes significant damage to the cells and skin, bone marrow, and digestive tract. When radiographing animals, lead aprons, gloves, thyroid collars, and glasses should be worn. Never place any body part, whether lead-covered or not, directly in the primary beam. All persons not essential to taking the X-ray should be cleared from the room. The collimator should be used to restrict the primary beam to the absolute minimum needed to take an adequate X-ray and to minimize scatter. A dosimetry badge should be worn whenever an X-ray is

taken to measure the amount of cumulative radiation exposure to team members.

Veterinary Technician

Veterinary technicians or technologists are usually those employees who have obtained an associate's or bachelor's degree from an AVMA-accredited program. A technician's role in the veterinary clinic is essential to the smooth operation of the clinic and to assist veterinarians in performance of their tasks. The duties of a technician encompass most of the tasks performed in the practice except those that can only be performed by the veterinarian, such as performing surgery, prescribing drugs, and making a diagnosis. Technical tasks should be performed by the technician to free up the veterinarian to perform tasks that can only be done by graduates of veterinary colleges. The well-trained technician or technologist can provide great leverage in a veterinary clinic and boost financial profits if allowed to do the tasks he or she is trained to perform.

Customer Service

Veterinary practices that are successful in today's competitive environment practice exceptional client service. The goals of exceptional client service are fairly straightforward. The practice strives to create a personal bond with the client and maintain an honest, courteous open policy when dealing with clients. The practice also strives to create a pleasant environment for clients and staff. The buildings are odor-free, clean, and tastefully decorated. These practices hire and maintain a well-trained, personable staff and serve the human-animal bond. Most importantly, they put the client and patient first by providing the services they want in a cost-effective manner. All these strategies build towards one goal: brand loyalty that creates a long-term client relationship.

Developing Client Relationships

Building a long-standing client relationship takes teamwork. All team members in a veterinary practice must be committed to providing the highest possible level of client service. It is essential that staff is well trained in customer service because up to 80% of the client visit is spent with staff members other than the doctor. A professional image should be projected. This includes excellent appearance, manners, grammar, and knowledge about medical and surgical procedures. All team members should display a positive attitude, courtesy, manners, and compassion. Excellent communication skills are essential as well, whether talking to perspective new clients on the phone, dealing with upset clients, or explaining the results of procedures or tests to the client.

Marketing Strategies

Marketing a veterinary clinic consists of a number of factors designed to foster the image the clinic wishes to project to the public while keeping in mind cost-effectiveness. Newsletters are an excellent means to promote a quality image, to bond clients to the practice, to inform clients of staff changes, and to educate clients. Client handouts (fact sheets) are one-page, to-the-point, easy to comprehend sheets on any medical condition. They are excellent client education tools and can also be distributed to local media if they contact your client regarding timely news stories about particular diseases. The clinic can also sponsor special events such as contests, classes, or an open house to allow clients and the public a glimpse into the world of veterinary medicine and what the practice has to offer to the community.

Employee Termination

The laws that govern employee termination are designed to protect the employee because of the perceived inequality of bargaining power between employer and employee. Employers should keep employee records and should also have a policy statement, an employee handbook, and copies of all employee performance appraisals. The federal government and state government all have employment laws. Federal employment law is considered to be the minimum requirement; state law may exceed federal employment law. When terminating an employment relationship, the employer should have an accurate job description and records of unacceptable job performance. Discuss the employee's poor performance and give them an opportunity to improve. If the employee fails to comply with the terms of the discussion, draft a written notice about the unacceptable behavior and have the employee initial the notice. Keep records of all the written notices in a safe and secure place.

Prescriptions

A prescription must be written by a veterinarian. The following items need to be included on a written prescription: the veterinarian's name and address, and in the case of a controlled substance, the veterinarian's phone number and DEA number, the client's name and address, the name, species, age, weight, and breed of the animal being prescribed the medicine, the name of the drug (either generic name or trade name), the dose, refill instructions and dosing instructions, the date the prescription was written, and the veterinarian's signature. Only a veterinarian can prescribe drugs. Staff members should not refill or issue medications without the prior approval of the veterinarian.

Controlled Substances

Controlled substances are highly regulated and special rules govern their use. Schedule I drugs have no accepted medical use in the United States. Examples of these drugs include heroin and LSD. Schedule II drugs have a high potential for abuse and include narcotic, depressant, and stimulant drugs, such as morphine or pentobarbital. Abuse of Schedule II drugs can lead to severe dependence, and they cannot be refilled. Examples of Schedule III drugs include Tylenol with codeine and anabolic steroids. Schedule IV drugs include diazepam and phenobarbital. Schedule V drugs include Robitussin AC or Lomotil. Schedule III, IV, and V drugs can be refilled a maximum of five times in a six-month time period.

Job Discrimination

Federal Equal Employment Opportunity (EEO) laws prohibit job discrimination. These are based upon the following laws: Title VII of the Civil Rights Act of 1964 prohibits employment discrimination based upon race, color, religion, sex, or national origin, the Age Discrimination in Employment Act of 1967 protects workers who are over the age of 40, the Equal Pay Act of 1963 prohibits sex-based wage discrimination. In addition, Title I and Title V of the Americans with Disabilities Act of 1990 prohibit employment discrimination against people that have disabilities, the Civil Rights Act of 1991 provides monetary damages in cases of intentional employment discrimination, and Sections 501 and 505 of the Rehabilitation Act of 1973 prohibit the discrimination against qualified federal workers with disabilities.

Sexual Harassment

Sexual harassment falls under Title VII of the Civil Rights Act of 1964. This act applies to employers with 15 or more employees. Sexual harassment consists of unwelcome sexual advances or request for sexual acts. It also includes sexual verbal and physical conduct. This harassment also interferes with a person's work performance, creates a hostile, intimidating, or offensive workplace, or affects the person's employment. The main feature is that the conduct must be unwelcome. Both sexes can be the harasser, and the harasser can be anyone the person has contact with in the workplace. It also includes all those who are affected by the harasser's conduct. The unwelcome conduct may happen without economic injury or discharge of the victim.

Malpractice

To win a case of veterinary malpractice, a number of factors must be proven. The veterinarian must be under a duty of care, or have accepted the responsibility to treat the animal when the owner brought the animal to the clinic. The veterinarian's treatment or lack of treatment must have fallen below

the professional standard of care, which is established through expert testimony. The plaintiff then needs to prove that this below-standard care was the proximate cause of the animal's injury. The last factor that must be proved is that the harm inflicted by the defendant resulted in damages (primarily monetary) to the plaintiff (the owner) because he or she is the party bringing suit, not the pet.

In order for a client to win a malpractice case, the client (or lawyer) must show proof that the veterinarian was negligent. Meticulous record-keeping is a must in order to avoid a malpractice case. Some points to keep in mind to avoid legal problems are as follows. Clients can assist with handling their pets, but the veterinarian will be held responsible if the client is injured in the process. Good record-keeping includes keeping records of treatments, medications, client instructions, and vaccinations. Do not release an animal from the clinic until it is fully awake if it was anesthetized or given a sedative/tranquilizer. Obtain written authorization from the client prior to any surgical procedure or before the animal is euthanized. Keep radiographs, laboratory results, and medical records because they are the property of the veterinary hospital not the client; clients can have copies.

Suspected Animal Abuse

Veterinarians may have an ethical responsibility in animal abuse cases, but legally, state laws vary; the majority of the states do not have any formal laws for reporting animal abuse. Some states have what are considered good Samaritan laws that encourage the veterinarian or veterinary staff to report cases of animal abuse without the fear of breaching client expectations of confidentiality or being opened up to legal recourse. Abuse can be difficult to define, and states vary as to what type of abuse is considered criminal. Some states encourage voluntary reporting, while a few states have mandatory reporting laws. The American Veterinary Medical Association's stance is as follows: "When these situations cannot be resolved through (client) education, the AVMA considers it the responsibility of the veterinarian to report such cases to appropriate authorities."

Animal Companionship

Studies that have been undertaken regarding the human-animal bond have found that pet ownership and/or companionship provides numerous physical and mental health benefits to a person's life. Pets give people attention and attract attention to people who may not receive much notice from others. This decreases loneliness, increases human to human interaction, and gives the person a sense of well-being. With children, the pet gives the child a chance to

experience responsibility and gives an opportunity to learn to nurture. Owning a pet often encourages exercise, and their antics often provide comic relief, which can decrease stress levels. Many of the benefits of the human-animal bond have been proven to lower blood pressure and lessen the need for medication, particularly in the elderly.

Species

Feline and Canine

Abdominal Incision Sutures

For abdominal incision closure in the dog and cat, the following suture size, material, pattern, and placement is recommended. For the closure of the linea alba (rectus abdominis muscle), the appropriate suture material is nylon, polyglyconate, polydioxanone, or polypropylene. Depending on the size of the animal, dogs require 2-0 to 1 suture size, while in cats, 3-0 to 2-0 are the proper size. The incision is closed using a simple continuous suture pattern at 3- to 12-mm placement depending on the size of the animal. The second layer to be closed, the subcutaneous layer, should be closed in a simple continuous pattern using a synthetic absorbable suture of 4-0 to 3-0 in size. The suture interval should be 3 to 12 mm apart. The skin is closed with nylon or polypropylene suture from 3-0 to 2-0 in size in a cruciate or simple interrupted pattern spaced 5 to 10 mm apart.

Cellulitis

Dogs and cats frequently present with abscesses and cellulitis primarily due to fighting bites and scratches. Cats are especially prone to deep infections. Other causes include plant awns, bullet wounds, or skin punctures. There are numerous bacteria present in these wounds such as anaerobes, *Streptococci* , and *Pasteurella* . The signs vary but frequently occur on the legs, head, back, and the tail base. Carefully palpation will reveal the painful lesion. The animal may have a fever and the surrounding lymph nodes may also be swollen. Treatment involves draining and irrigation of the abscess, antibiotics to treat any concurrent cellulitis, and possibly pain medication to relieve pain and inflammation. Rabies vaccination status should also be updated at the same time. Recurring abscesses in cats may indicate an immune suppression disease such as feline immunodeficiency disease or feline leukemia.

Thoracocentesis

Thoracocentesis is performed to remove fluid or air from the chest cavity or to collect a fluid sample for histopathology. A butterfly catheter attached to a 3-way stopcock to a syringe is used. If this is being performed on a large dog, the needle should be held in a horizontal position to fully penetrate through the intercostal muscles. In smaller animals, the needle should be held at an oblique angle to the pleural space to minimize the chance of lacerating the lung. The

operator holds the “wings” of the catheter and advances the needle into the prepared site until the needle is in the pleural space. Then, the operator releases the wings, as a “loose” needle is less likely to lacerate lung tissue when the animal breathes or if it struggles. The stopcock is then used to evacuate fluid or air.

Diabetes Insipidus

Diabetes insipidus is a disease characterized by water imbalance in the body. It is a rare disease in dogs and cats and can be categorized into two types: Central diabetes insipidus is caused by a deficiency in antidiuretic hormone due to a pituitary tumor; nephrogenic diabetes insipidus is caused by the kidney not responding to antidiuretic hormone. An animal can be born with either of these two types of diabetes insipidus or it can acquire diabetes insipidus later in life due to drug toxicity, trauma, other disease conditions, or neoplasia. The clinical signs of diabetes insipidus are increased drinking and urination and a low specific gravity of the urine (<1.008). Treatment for central diabetes insipidus is with desmopressin, given either as drops in the eye conjunctiva or intranasally. Nephrogenic diabetes insipidus is treated with oral chlorothiazide.

Diabetes Mellitus

Treatment of diabetes mellitus in the dog and cat involves a combination of medication, diet modification, and consistent levels of exercise. A newly diagnosed animal may need hospitalization to stabilize health and to titrate insulin supplementation. Dogs typically need twice-daily injections of insulin. Cats may be treated with strict diet modification (high-protein diet), oral glipizide (a hypoglycemic agent), or with insulin injections. In all animals, soft, moist food should be avoided due to its hyperglycemic effect after eating. Obese animals should gradually lose weight; nonobese pets should maintain weight and eat a consistent kibble diet. Diets should be high-fiber, high in complex carbohydrates, and low in fat. Exercise should be encouraged on a consistent schedule each day. Clients should monitor their pet’s urine for glucosuria and body weight. The animal should be brought in for a blood glucose curve on a regular basis.

Walking Dandruff

Walking dandruff is the common name given to the disease condition known as cheyletiellosis. This parasite affects cats, dogs, and rabbits and can cause human lesions as well. *Cheyletiella* mites are large mites that cause excessive skin scaling, giving it the name walking dandruff. The mite is highly contagious and is spread through the environment and through direct contact. The common clinical sign is scaling or dandruff over the dorsum. Itching may or may not be

present and some cats can act bizarrely and exhibit excessive grooming and hair loss. All animals in the household must undergo treatment. Topical medications (shampooing) need to be given weekly for one to two months, along with rinses of lime-sulfur and pyrethrins. Organophosphates can be used in dogs but not in other species. Ivermectin injections can also be given as three doses in two-week intervals.

Formation of Heinz Bodies

Regenerative hemolytic anemia is caused by formation of Heinz bodies, or erythrocyte inclusions composed of denatured and oxidized hemoglobin. This condition occurs after animals feed on chemical or dietary substances that oxidize hemoglobin such as acetaminophen, onions, zinc, or propylene glycol. Clinical signs include anorexia, paleness of the mucous membranes, cyanosis (if methemoglobinemia has occurred), weakness, fever, and blood in the urine. Blood work reveals the presence of a regenerative anemia, or an increased number of polychromatophils or young red blood cells, and Heinz bodies, which appear as pale, round, protruding inclusions. New methylene blue staining rapidly reveals the Heinz bodies. Normal cats have a few (<5%) Heinz bodies. Treatment is aimed at removal of the toxic substance and supportive care until the body recovers from the assault.

Red Blood Cell Cycle

Red blood cells are produced in the bone marrow in response to kidney release of erythropoietin (EPO), which also stimulates platelet production. In the dog, RBCs have a life of about one hundred days; cats turn over RBCs every 85 days. The normal morphology of the dog's RBC is a biconcave disc featuring a prominent central pallor. Cats have RBCs shaped as a biconcave disc but with a smaller area of central pallor. The RBCs are composed of hemoglobin proteins that carry blood gases and a cell membrane. Their main function is to carry oxygen to the body tissues and to take carbon dioxide away from body tissues and exchange it for oxygen in the lung. Metabolic pathways in the RBC include anaerobic glycolysis to produce energy needed by the cell and pathways to protect hemoglobin from oxidation, such as methemoglobin reductase and the hexose monophosphate shunt.

Immune-Mediated Hemolytic Anemia

Immune-mediated hemolytic anemia occurs when red blood cells (RBCs) become coated with antibodies as they circulate throughout the body. The antibodies can be classified as warm type, or those that are reactive at body temperatures (IgG) or cold type, or those that are reactive below normal body temperature (IgM). This antibody coating accelerates the destruction or

removal of the RBCs to such a degree the body becomes depleted of circulating RBCs. This antibody coating occurs due to exposure to antigens, adsorption of antigen-antibody complexes, or disease-producing organisms. Causes include lymphoma, systemic lupus erythematosus, drugs, heartworm disease, viruses (feline leukemia virus), *Ehrlichia* , leptospirosis, *Babesia* , hemobartonella, neonatal isoerythrolysis, immune system dysfunction, or Type III hypersensitivity reaction (Arthrus reaction). Some cases are idiopathic without any known cause.

Oral Tumors

Many cases of oral tumors in the dog or cat need to be treated with extensive surgical resection. Maxillectomy or mandibulectomy can be complete or partial; both are indicated to cure those tumors that have not metastasized or they can prolong the life of those animals affected by tumors that have already metastasized. Depending on the site of the tumor, various portions of bones of the face or jaw are removed in order to provide a wide margin (one to two centimeters surrounding the tumor tissue) to ensure that all or the bulk of tumor cells are removed from the animal. The surgeon must be careful when performing the excision, suturing, and handling the tumor to avoid seeding tumor cells into tumor-free tissue.

Feline-Specific

Catheterizing a Male Cat

Male cats frequently will need catheterization to relieve urinary tract blockages. A 3 ½-Fr size catheter is frequently used in this situation. The cat is sedated and laid on its side. The penis is extruded from the prepuce and held in a parallel line to the vertebral column. The penis and prepuce should be cleansed with an antiseptic solution. The catheter is attached to a syringe with sterile saline. The tip of the catheter should be lubricated with an aqueous lubricant to prevent blockage of the catheter tip. The catheter should be slowly advanced into the urethra. Depending on where the blockage is located, the catheter may stop while it is being advanced. The saline should be infused slowly as the catheter is advanced to dislodge the obstruction. Once the catheter reaches the bladder, urine should flow and the syringe should be removed to facilitate bladder evacuation.

Hyperthyroidism

A cat with hyperthyroidism can be treated medically, with surgery, or with radioactive-iodine therapy. Most animals are treated medically. Methimazole (Tapazole) is commonly used at dosages of 15 mg per kg twice daily. It acts by reducing the production and the release of thyroid hormone from the thyroid

gland. Some side effects include anorexia, lethargy, vomiting, fever, and anemia. Surgery can sometimes be performed to remove the thyroid gland (thyroidectomy). If surgery is performed, the surgeon has to be careful not to damage the parathyroid glands. Radioactive iodine therapy is an effective treatment. The radioactive iodine is injected and taken up by the thyroid gland, which destroys the abnormal thyroid tissue. Because of the lack of therapy centers, the cat must remain in the facility for approximately two weeks until radioactivity levels fall to safe levels.

Toxoplasma Gondii

The enteroepithelial life cycle of *Toxoplasma gondii* is only found in the cat, which is the definitive host for this organism. This life cycle from time of ingestion to the formation of infective oocytes can take as little as three days. The cat ingests tissues carrying the organism in cysts. The cyst walls are dissolved by the intestinal enzymes and release bradyzoites, which penetrate the small intestinal epithelial cells. Here, schizonts release merozoites to form female and male gamonts. The gamonts form macrogamonts, which are then formed into an oocyst, and are passed in the feces. When the oocysts are exposed to air and moisture, they sporulate within five days. The oocysts have two sporocysts, each having four sporozoites.

Ringworm

Examination for ringworm can be aided by using a Wood's lamp, which may causes hair shafts infected with *Microsporum canis* to glow an apple-green color; however, because this glow may have other causes, the cat's skin should be carefully examined and a sample should be taken for fungal culture. Using a new, clean toothbrush to aggressively comb over the body and paying close attention to the inside of the ears, the face, and the feet provide a good representative sample. The toothbrush bristles should be stabbed into the fungal growth media. In positive cats, treatment strategies vary. Positive cats should be separated from other cats until treated. Many short-haired cats spontaneously overcome the disease. Other cats can be treated with oral griseofulvin, ketoconazole, itraconazole, lime-sulfur dips, or miconazole shampoos.

Osteoarthritis

Osteoarthritis is the pathological change of the diarthrodial synovial articulation. The disease process includes deterioration of articular cartilage, changes in the soft tissues, low-grade inflammation, osteophyte formation, and remodeling of the bones. Signs of osteoarthritis in the cat can be subtle, but it is an important cause of decreased quality of life in older cats. These signs include lack of attention to grooming, a reluctance or inability to jump up to higher

surfaces, litter box issues (defecating or urinating outside of the litter box), hiding, increased time sleeping, and pain caused by or avoidance of brushing or petting. Treatment of osteoarthritis in cats includes use of nonsteroidal anti-inflammatory agents. This can pose a problem with cats, as most NSAIDs have long half lives in cats. Meloxicam or ketoprofen have been used with success to relieve the pain and inflammation in cats with osteoarthritis.

Cat Acne

Kitty acne presents as reddened papules, comedones, and crusting on the cat's chin and occasionally around the mouth. Some cats are prone to develop acne due to immune issues, increased sebum secretion, or keratinization abnormalities. Poor grooming can also predispose a cat to developing cat acne. Occasionally, the disease becomes more severe with pain, nodule formation, pustules, and hair loss, indicating the cat has developed furunculosis. Diagnosis is based upon clinical signs. Occasionally, biopsies are taken if there are questions about another cause of the clinical signs; this includes demodicosis, neoplasia, or dermatophytosis. Treatment for a mild case includes gently scraping off crusts, shampooing once a week with an antiseborrheic shampoo, and cleansing with benzoyl peroxide. More severe cases may require systemic antibiotics.

Nutritional Needs

Cats are carnivorous animals and derive most of their protein from fish, meat, and other animal products. Cats need the amino acid taurine in the diet. A deficiency of taurine causes a number of disorders: cardiomyopathy, retinal degeneration and blindness, reproductive failure, congenital defects, deafness, and impaired immunological response. Fatty acids (omega-3 and omega-6) are essential in the cat's diet. Deficiencies of fatty acids cause nervous system disorders. Vitamin deficiency in the cat can cause a variety of health disorders. Because cats are unable to synthesize some vitamins from vitamin precursors, they need to ingest these in the diet. Vitamin A deficiency can lead to eye disorders. Large doses of vitamins can also damage the health of the cat. Too much vitamin A (as occurs from feeding liver) can cause hypervitaminosis A in the kitten, which produces skeletal lesions.

Eosinophilic Granulomas Complex

The three diseases are indolent ulcer, eosinophilic plaque, and eosinophilic granulomas. In the clinic, impression smears should be taken of the active lesions; these smears may reveal a large number of eosinophils. In order to rule out other similar-appearing diseases, different tests may be performed, including allergy testing through intradermal skin testing, food elimination trials, or elimination of fleas to rule out flea bite hypersensitivity. To

differentiate the three diseases, a biopsy of the lesions should be taken and examined at a diagnostic laboratory. Treatment of the three diseases revolves around use of corticosteroids; either through use of injectable preparations or through oral administration. In some cases, immunosuppressive agents and antibiotics may be used.

Elimination Problems

Elimination problems are the most common behavior problem in cats. Most involve urination, with spraying being a top concern. Male cats are the primary culprits, especially those that live in a multi-cat household with at least one female cat, although female cats may spray. Diagnosis is based upon observation and history. The workup for spraying includes a physical exam, blood testing, and urinalysis. Medical problems should be corrected before a cat is labeled a urine sprayer. Treatment begins with neutering intact cats, manipulating the environment such as decreasing access to windows and doors, decreasing cat household numbers, and making the sprayed areas less attractive to the cat. Behavior-altering drugs can also be used to reduce the cat's anxiety levels. Medications used include benzodiazepines, tricyclic antidepressants, selective serotonin re-uptake inhibitors, buspirone, progestins, antihistamines, and pheromones.

Asthma

Feline asthma is a disease that causes bronchoconstriction in the cat's lungs. The signs exhibited by a cat with asthma include wheezing and coughing. When the lungs are examined with a stethoscope, adventitious sounds (crackles) are frequently heard. Common tests for a cat with asthma include fecal examination to look for lung parasites, heartworm testing, and a complete blood count, which frequently reveals an increased number of eosinophils. Radiology of the lungs may be normal, but with more severe cases, the lungs reveal an inflammatory airway with prominent bronchial markings; occasionally, atelectasis (collapsed lung) will be seen. The primary treatment for cats with asthma revolves around judicious use of corticosteroids (prednisone, methylprednisolone) that may be combined with terbutaline or theophylline. If testing reveals parasites, treatment should also be given to eliminate the organism.

Review Video: Asthma and Allergens
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Aggression

Cats can display aggression for a number of reasons. Cats that have not been properly socialized to humans before the age of 12 weeks will be fearful and/or aggressive towards humans. The ideal time to socialize kittens is between two and seven weeks of age. This can be accomplished by handling the kitten five minutes a day. Cats that are hand-raised by humans without interacting with other cats can exhibit play aggression. Fear and pain can also make a cat aggressive. Queens can display maternal aggression when the kittens are very young. Cats can also display predatory aggression, assertion aggression, territorial aggression, intercat aggression (male-male), or redirected aggression.

Acetaminophen Toxicity

Acetaminophen is a toxic substance to cats. The substance is readily absorbed from the gastrointestinal tract, usually within one hour after ingestion, unless stomach emptying is delayed by a high-carbohydrate meal, which may delay peak plasma levels for as long as four hours after swallowing. Cats have a more difficult time metabolizing the substance as they do not have the glucuronyl transferase necessary to conjugate acetaminophen; therefore, cats SHOULD NOT be given acetaminophen for any reason. Cats may also develop liver lesions and methemoglobinemia. A dose as low as 40 mg/kg causes symptoms. Signs of acetaminophen poisoning in cats include anorexia, vomiting, facial and paw edema, methemoglobinemia, respiratory distress, cyanosis, and weakness. Treatment goals include preventing the substance from being absorbed by the body by inducing vomiting and giving activated charcoal if recently ingested, giving N-acetylcysteine to bind metabolites, giving ascorbic acid, and providing supportive care, such as fluids and respiratory support.

FIV

Treatment for feline immunodeficiency virus (FIV) is primarily supportive and based upon treating the symptoms as they develop. Use of antiviral drugs such as interferon alpha may be of some benefit. Keeping the cat from being infected in the first place is the best approach to FIV. Because cat bites are the primary mode of transmission, cats should be kept indoors to reduce the possibility of contact with an infected cat. Before bringing a new cat into the house and introducing it to the resident cats, the new cat should be tested. Vaccination with an inactivated vaccine should be encouraged. Vaccinating cats which are FIV-negative three times three weeks apart will help protect against infection, although the vaccine is not totally protective. The vaccination gives a positive result on the screening tests.

Cats that test positive for feline immunodeficiency virus (FIV) need to be carefully managed to prevent the further spread of the virus and to optimize their health. They should be fed a balanced diet to make sure they get the

nutrition they need. Raw meats and dairy products are not recommended due to the possibility of contracting food-borne disease. Cats should be spayed or neutered and should have biannual health check-ups. Any change in condition must be carefully investigated and monitored. An FIV-positive cat should be a strictly indoor cat to minimize exposure to pathogens and to control the spread of the virus to other cats with which the FIV-positive cat may come in contact

FIP

Feline infectious peritonitis (FIP) is a contagious disease that is spread via the oronasal route (saliva), by an infected queen passing the virus to its kittens or through the feces. The most common route of transmission is from the mother to the kittens at about five to eight weeks of age. It is much more likely to occur in areas where cats are congregated such as catteries or in animal shelters. The most likely cats to contract the pathogenic coronavirus that causes FIP are cats with weak immune systems. This includes cats infected with the feline leukemia virus, kittens, and older cats. FIP usually develops in young cats (those from six months to five years of age) but it may cause disease in cats of all ages.

FeLV

Feline leukemia virus (FeLV) causes one of two syndromes to occur in persistently infected cats. The neoplastic forms include myeloid leukemia and lymphosarcoma and the non-neoplastic forms include kidney, reproductive, or immunosuppressive disease. The virus is spread by persistently infected cats through their secretions, especially saliva and nasal discharge. Infection can be spread via the environment such as common feeding or water bowls or litter boxes or via direct contact through bites, grooming, or from the mother to unborn and newborn kittens. The virus does not live long outside the body and most common household disinfectants kill the virus. In the United States, it is estimated that up to three percent of all cats are infected with the feline leukemia virus.

Feline leukemia virus (FeLV) infections can be prevented by monitoring the cat's infection status and keeping FeLV-negative cats away from cats that are infected or who have unknown infectious status. This is best accomplished by keeping cats indoors to prevent fighting and bite wounds. Prior to bringing a new cat into the household, it should be tested for FeLV. Vaccinations are available and should only be used in FeLV-negative cats; the vaccination does not interfere with testing results. Any cat that tests positive for FeLV should be kept separate from cats that test negative, should be spayed or neutered, and should be closely monitored with thorough medical examinations given twice-yearly or more frequently if necessary.

Biliary Tract Disease

Cats with cholangitis and cholangiohepatitis (inflammation of the biliary system) can exhibit a wide variety of clinical signs ranging from mild, acute, intermittent, or chronic. Owners will notice their cat refuses to eat, may vomit, lose weight, or be lethargic. Physical examination of these cats may reveal icterus, fever, and a swollen liver. Based upon these signs and the examination, some other diseases that should be ruled out include feline infectious peritonitis, liver cancer, fatty liver, feline leukemia virus, or other liver problems. Lab work-up will show an increase in the alanine aminotransferase (ALT), alkaline phosphatase (ALP), and gamma-glutamyl transferase (GGT) levels as well as increased levels of bilirubin in the blood and urine. To diagnose the disease, a liver biopsy should be taken. Treatment is with antibiotics, diet, and possibly surgery to relieve bile duct obstruction.

Panleukopenia Treatment

Treatment of a cat with feline panleukopenia primarily depends upon supportive care. It is important to provide rehydration and electrolytes to these cats as they will be severely dehydrated. If they are actively vomiting and have severe diarrhea, food should be withheld until these symptoms improve. Use of broad-spectrum antibiotics should be considered in order to combat secondary bacterial infections. With good nursing care and prompt diagnosis, many cats and kittens will recover; however, during the acute illness and for six months after recovery, the affected cat or kitten should remain inside, as they will shed the virus during this time frame. Most vaccination combinations carry the modified live vaccine for feline panleukopenia. Maternal antibodies last for up to three months in kittens from vaccinated queens. A series of three vaccinations commencing at six weeks of age and an annual booster provide superior protection against this disease.

Infectious Anemia

Feline infectious anemia (Haemobartonellosis) is caused by the rickettsia *Haemobartonella felis* , an obligate intracellular bacterium. It is usually an asymptomatic infection or it may lead to a regenerative anemia, particularly if the animal is stressed or has a concurrent disease. A cat with an acute occurrence of the disease will have a fever, be lethargic, lose its appetite, become jaundiced, and have an enlarged spleen. Regenerative anemia is seen upon blood testing; FeLV, FIP, and FIV have nonregenerative anemia. Blood smears also reveal the *H. felis* organism. Treatment will depend upon good supportive care, doxycycline or tetracycline for two to three weeks, and possibly prednisone if the anemia is severe. Because it is thought that arthropods or fleas are the vectors, cats should be treated for external parasites.

Lower Urinary Tract Disease

Feline lower urinary tract disease manifests as blood in the urine, straining when urinating, pollakiuria, urinating in inappropriate places, and partial or complete obstruction of the urethra. It is seen in both genders. A cat with this disorder has a thickened bladder wall when palpated, or if obstructed, a distended bladder. Sometimes crystal plugs can been found near the opening of the urethra. The most common stones in the cat are calcium oxalate, urate, and magnesium ammonium phosphate. Diagnosis is based upon signs, physical examination, and urinalysis. Urinalysis reveals crystal-type cells and frequently other cell types as well. Treatment is based upon relieving blockages, treating concurrent infections, and dissolution of uroliths by diet.

Insulin Types for Diabetes

Cats with diabetes mellitus are managed with oral hypoglycemic agents or insulin injections combined with dietary management. There are four main insulin types used in therapy for diabetic cats: Glargine is long-acting human recombinant insulin that was designed for human use; it has four amino acids that are different from cat insulin. Protamine zinc insulin is a type of insulin combined with zinc and with protamine, a protein derived from salmon testes. It also has four amino acids that are different from cat insulin. Vetsulin is the only officially approved insulin for cats and dogs. It is porcine zinc insulin that differs from cat insulin by three amino acids. Neutral Protamine Hagedorn (NPH) is human recombinant insulin with protamine and zinc. It differs from cat insulin by four amino acids.

Plague

Most domestic animals are resistant to plague with the exception of the cat. A cat most commonly becomes infected with plague through feeding on infected rodents. After initial infection, the cat incubates the bacteria for one to four days and then show signs of bubonic plague. The cat loses its appetite, has a fever, is depressed, and has a draining abscessed lymph node. Sometimes the cat also has ulcers in the mouth, eye discharge, and/or cellulitis. The organism is diagnosed by culturing the blood or tissues and by fluorescent antibody staining. Because this is a dangerous pathogen that humans can contract, precautions should be taken in areas endemic for plague if a cat displaying these symptoms is encountered. Treatment consists of lancing and draining abscesses and treating with antibiotics such as gentamicin, streptomycin, doxycycline, and chloramphenicol.

Hypertrophic Cardiomyopathy

Medical treatment for feline hypertrophic cardiomyopathy includes medications to help the heart and lungs function better and to prevent thromboembolism

from forming. Diltiazem helps improve diastolic relaxation, causes vasodilatation, inhibits platelets, stops arrhythmias, and slows the sinus rate and can also reduce heart muscle enlargement. Dosage is typically 10 mg/kg once a day or 7.5 to 15 mg every eight hours. Beta blockers (atenolol or propranolol) can be given as well to control sinus tachycardia, stop arrhythmias, inhibit platelets, and slow the sinus rhythm. Dosage is 6.25 to 12.5 mg once or twice daily for atenolol or 2.5 to 10 mg two or three times daily. Furosemide (1 to 2 mg/kg once to three times daily) controls pulmonary edema, but cats are sensitive to the drug. Aspirin therapy (80 mg every two to three days) may decrease thrombi risk. Nitroglycerin ointment can be used to stabilize cats with pulmonary complications.

Vaccine-Associated Sarcoma

Vaccine-associated sarcoma (fibrosarcoma) can occur in cats at injection sites. This is an invasive sarcoma; the tumor can invade the surrounding tissues and extensive surgery may be required to completely remove the cancerous tissue. It is though that an adjuvant in vaccines, aluminum hydroxide, is responsible for initiation of sarcoma formation. Surgery followed by radiation therapy is the recommended therapy. Complete removal of the tumor plus at least a 2-cm margin of normal tissue is recommended. Some cases call for amputation of the limb if the tumor is located on a limb. To minimize the risk of a sarcoma, it is recommended that cats receive injections of rabies and feline leukemia vaccine in the rear legs; rabies vaccine in the right rear and leukemia vaccine in the left rear leg.

Canine Specific

Oral Melanosarcoma

Melanosarcoma is the most common oral tumor in dogs and the third most common oral tumor in the cat. The tumor begins in the gingival tissue and is locally invasive. It also metastasizes early in the disease course; by the time it is diagnosed, it has already metastasized. It is commonly seen in older dogs that have oral mucosa that is darkly pigmented. It appears as a dark or light gray oral growth. The dog may have halitosis, salivate excessively, have difficulty eating, have loose teeth around the tumor site, and may lose weight. Other disease conditions to rule out include dental abscess, ameloblastoma, epilus, or other oral tumors. A deep-tissue biopsy is needed to diagnose the tumor. Surgery, such as hemimaxillectomy or hemimandibulectomy, is required to remove the growth. Some animals will also benefit from radiation and chemotherapy postoperatively.

Gastric Dilation and Volvulus Syndrome

Treatment for gastric dilation and volvulus syndrome needs to begin as soon as the animal is presented for treatment and diagnosis is made. IV fluids should be started and the dog should have supplemental oxygen. A stomach tube should be passed to attempt gastric decompression. A light sedative should be used to facilitate passage. The animal should be prepared for an exploratory celiotomy to derotate the stomach and to perform a right-sided gastropexy. The abdominal organs should be examined for signs of compromise or necrosis. Portions of the stomach wall or the entire spleen may need to be removed. The animal should remain hospitalized following surgery and activity should be severely restricted for two weeks after surgery. Supportive IV fluids, corticosteroids, and broad-spectrum antibiotics are warranted to stabilize the animal postsurgery. The diet should be modified after surgery and may need to be permanently changed.

Hygroma Forms

Hygromas form after repeated trauma to the soft tissue that overlies bony prominences on a dog's body. These swellings contain serous fluid and are common in young, large-breed dogs housed on hard surfaces. Pressure on the tissues leads to a decrease in vascular supply, which causes ischemia and edema. The collection of edematous fluid incites an inflammatory reaction and a thick capsule of fibrous connective tissue lined with granulation tissue forms. The most effective treatments are removal from hard surfaces and/or padding. Surgery is only indicated in painful lesions, lesions that chronically drain, or those that are chronically infected. If surgery is performed, the hygroma must be carefully dissected and the wound closed.

Mammary Tumors

Treatment of mammary tumors in the female dog, whether the tumor is benign or malignant, consists of surgical removal. Depending on the type of tumor present, the options include a lumpectomy, which entails the removal of the gland with the tumor as well as a 1-cm margin of surrounding normal-appearing tissue. A mastectomy can be performed in which the tumor plus the associated gland is removed. A regional mastectomy consists of removal of the tumor and the associated gland plus its associated vascular and lymphatic drainage system and lymph node. In a unilateral mastectomy, the tumor, its mammary gland, and all the mammary glands on the same side of the body plus the vascular and lymphatic drainage system and lymph node are removed. A bilateral mastectomy removes the same structures as the unilateral mastectomy but on both sides of the body.

Stomach Tubes

A stomach tube is passed in the dog for a number of reasons: to relieve bloat from gastric dilatation volvulus, to empty and lavage the stomach, and to administer food or barium. The length and the diameter of the tube should match the size of the dog. The tube should be measured from the tip of the nose to the ninth intercostal space to make sure the tube enters the stomach. Intubation to relieve bloat or to give barium is performed on the dog when it is awake; sedation is needed if the dog has ingested poison. A speculum is placed in the dog's mouth as it is held in sternal recumbency; the tube end is lubricated and introduced into the oral cavity and advanced into the esophagus. The tube may meet some resistance at the cardia, but as it enters the stomach, air or fluid will flow out.

Perineal Region Tumors

Tumors of the perianal region include anal sac carcinoma, or a malignant tumor of the apocrine glands of the anal sac, and perianal gland tumors, which are tumors of the modified sebaceous glands in the skin. Female dogs are predisposed to developing anal sac carcinoma; male dogs are more likely to suffer from perianal gland tumors. Dogs older than eight years of age are more likely to have these tumors. Due to gender prevalence, it is though that hormones influence the development of these tumors. In both cases, dogs present with a tumor mass in the perianal region. They may also lick or chew at the mass and scoot along the ground. Some masses make defecation difficult.

Avascular Necrosis of Femoral Head

Avascular necrosis of the femoral head (Legg-Calve-Perthes Disease) occurs when the blood supply to the head of the femur bone is damaged. The lack of blood supply causes the bone to degenerate and collapse and the cartilage on the femoral head become thickened and deformed. This causes inflammation and arthritis of the hip joint, leading to pain, limping, and atrophy of the thigh muscles of the affected leg; this usually occurs only in one leg. Both sexes are equally affected, and it is more common in the toy and terrier breeds, such as toy poodles, miniature pinchers, Manchester terriers, Cairn terriers, and West Highland white terriers. Young dogs (three to twelve months of age) are most commonly affected.

Avascular necrosis of the femoral head causes a gradual onset of lameness over a period of months. There is no history of trauma and the dog may chew at the affected hip or become irritable. When the joint is moved, it may cause pain and a crepitus may be felt when manipulating the joint. The muscles of the affected leg may atrophy, and the dog may occasionally carry the leg. Radiographs of the leg may reveal the following: flattening of the femoral head, widening of the joint space, and increased bone density of the femoral head and bone spurs.

Other diseases to consider when diagnosing avascular necrosis of the femoral head include traumatic injury to the hip and medial patellar luxation.

Medical treatment of avascular necrosis of the femoral head consists of strict cage rest and use of nonsteroidal anti-inflammatory agents along with physical therapy. This can only be successful in early stages if the femoral head has retained its normal shape and if it remains tightly seated in the hip socket. Usually the disease is not recognized or diagnosed early enough for conservative treatment to be effective. Surgery to excise the femoral head and neck along with immediate postsurgery physical therapy is the usual treatment. After the head and neck of the femur have been exposed, the femoral head is subluxated. The round ligament is severed and the femoral head is fully exposed. The neck and head are completely excised and the leg is returned to the normal position and the incision closed.

Patellar Luxation

Dogs with patellar luxation can either be medically or surgically treated. Animals with Grade 1 and Grade 2 luxation are typically treated medically with pain medication and rest. Some cases resolve when the patella stabilizes in the reduced position. In dogs requiring surgery, several options are available to treat the luxation. The femoral groove can be deepened to allow the knee cap to ride in its normal position. The soft tissues surrounding the knee cap can be reconstructed to provide stability to the patella. The tibial crest can be transposed to realign the patella, its tendon, and the quadriceps muscle. In some cases of deformities of the leg bones, the bones must be surgically reshaped. The type of procedure to be performed is decided on a case-by-case basis.

Hip Dysplasia

To perform an Orthopedic Foundation for America (OFA) radiograph certification for hip dysplasia, the dog is sedated and radiographs are taken when it is in dorsal recumbency. The rear limbs are extended and are parallel to each other. The knees are then rotated internally and the pelvis is symmetric. On the radiograph film, there must be permanent identification with the animal's name or registration number, the veterinarian's or hospital name, and the date the radiograph was taken. Along with the radiograph, the owner needs to complete and sign the OFA application. The radiographs of dogs, 2 years of age or older, are randomly evaluated by three board-certified veterinary radiologists. These radiologists evaluate nine anatomic hip areas: craniolateral acetabular rim, cranial acetabular margin, fovea capitus, femoral head, caudal acetabular rim, acetabular notch, dorsal acetabular margin, trochanteric fossa, and the junction of the femoral head and neck.

The Orthopedic Foundation of America (OFA) classifies hip dysplasia into seven phenotype classifications: excellent, good, fair, borderline, mild, moderate, or severe. Excellent indicated that the hip has a deep-seated femoral head that fits tightly into the acetabulum with minimal joint space. Good indicates that the femoral head fits well in the acetabulum but is slightly less than excellent. Fair indicates that the femoral head slightly slips out of the acetabulum. The dorsal acetabular rim may deviate in slightly. Borderline hips should be re-radiographed in six months. Mild hip dysplasia indicates that there is significant subluxation of the hips. Moderate hip dysplasia indicates that there is significant subluxation present, the femoral head is barely seated into a shallow acetabulum, and there are arthritic bone changes and sclerosis. In severe hip dysplasia, the hip has marked dysplasia; the femoral head is luxated out of the socket and there is marked arthritic bone changes.

Total Hip Replacement

A total hip replacement is done on mature dogs with severe arthritic changes that do not respond to medical management. When a hip is replaced, the femoral head is removed with a bone saw and the femur and pelvis are prepared for implant placement. The metal ball is made of titanium or cobalt-chromium. The hip socket, or acetabulum, is then replaced with a plastic cup. There are two procedures used to replace hips in dogs. One type uses bone cement to hold the new ball joint; the cement is injected into the acetabulum and the femur. The other type does not use cement. Instead, the stem of the ball is pushed down into the femur and new bone growth holds the stem in place.

Triple Pelvic Osteotomy

A triple pelvic osteotomy can be done on dogs with hip dysplasia to make the femoral head and acetabulum congruent. It works best on large-breed dogs when they are between eight and ten months of age. At this age, the bone is mature and can hold screws and implants, but the bone will still continue to grow and undergo acetabular remodeling after the surgery is completed. The bone is cut at three places: the pubis, the ischium, and the shaft of the ilium. The pelvis is then rotated to the desired position to improve coverage of the femoral head by the acetabulum and then a plate and screws are used to hold the joints in place.

Collecting Urine Sample

Urine can be collected by one of four procedures in a dog. If the animal passes urine on the exam table, this can be collected, but it is most likely to be highly contaminated with bacteria. The urine can still be tested for other indicators of a urinary tract infection, such as cells, protein, blood, and crystals. A mid-stream sample of urine can be collected while the dog is urinating. This sample

may still have contamination of bacteria from the lower urinary tract system, but it is a common practice. The bladder can be catheterized; this sample is considered to be unlikely to be contaminated. The ideal collection method is via a cystocentesis where a needle is passed through the abdominal wall directly into the bladder. Occasionally, blood from the needlestick will contaminate the sample.

Urinary Tract Infections

While the majority of cases of urinary tract infection in the dog are caused by a simple bacterial infection, with *Escherichia coli* being the primary pathogen, other conditions can exist that cause urinary tract infections. These should be considered in dogs with recurrent infections or infections that do not respond well to treatment. Pyelonephritis, or kidney infection, can be a complication of a neglected bladder infection. Bladder stones can be caused by the infection or they can be a cause of infection. The urachal diverticulum can cause repeated bladder infections until it is surgically removed. Bladder tumors, most commonly transitional cell carcinoma, can become infected and cause a bloody urine sample. Prostatitis in the un-neutered male dog can cause bladder infection; this infection will usually persist until the dog is neutered.

Gastric Ulcers

Gastric ulcers in dogs are caused by the breakdown of the gastric mucosal barrier. The normal or increased amounts of hydrochloric acid and pepsin in the stomach only make this breakdown worse. There are many causes of gastric ulcers in the dogs: drugs (nonsteroidal anti-inflammatory agents and corticosteroids), systemic disease (renal or liver disease, pancreatic disease), cancers (mastocytosis, gastrinoma), chronic gastritis, inflammatory bowel disease, *Helicobacter* infection, or pyloric outlet blockage. Some animals with gastric ulcers will appear healthy, while others may vomit blood, have pale mucous membranes, or blood in the stool. Treatment for gastric ulcers includes the use of mucosal protectants (sucralfate), histamine H2 receptor antagonists, and proton pump inhibitors.

Zinc Deficiency

Zinc is necessary for normal metabolism of proteins, nucleic acids, and membranes. One of the first signs of a zinc deficiency is skin alterations. Zinc-responsive dermatosis has histological lesions of marked parakeratosis with acanthosis and orthokeratotic hyperkeratosis. The dermis may have a moderate perivascular mixed inflammatory cell infiltrate. A dog that is zinc deficient loses hair, has adherent scales and crusts around the face and mucocutaneous junction, and has fissures and cracks in the foot pads. Other signs of deficiency include chronic diarrhea, growth retardation, thymic atrophy, lymphoid

depletion, hypogonadism, and changes in taste. Dogs can suffer a zinc deficiency from low-quality foods and puppies that are over-supplemented with minerals can have a disease similar to zinc deficiency. Alaskan malamutes and Siberian huskies can have a reduced ability to absorb zinc from the diet and display signs of a zinc-responsive dermatosis if not supplemented.

Treating Cherry Eye

Treatment for cherry eye in the dog consists of the use of topical anti-inflammatory medications and surgical replacement of the gland. Removal of the gland should be avoided, as the gland produces around half of the tear film and removal can predispose the dog to dry eye. Large-breed dogs affected with cherry eye can also have third eyelid cartilage eversion. A Morgan technique (or a modification of this technique) can be used to replace the gland. In the Morgan technique, an elliptical incision is made above and below the gland. The gland is then invaginated. The modified technique calls for excision of the conjunctiva over the gland; the gland is then invaginated. The incision is closed with buried 6-0 absorbable suture in a continuous pattern. An Elizabethan collar should be used postsurgery. Recurrence of the prolapse is a possibility and owners should be encouraged to repeat the surgery rather than remove the gland.

Ectropion

A dog with ectropion has everted eyelid margins that expose the palpebral conjunctiva. This exposes the eyeball to the environment and causes poor tear distribution, which can predispose the affected animal to chronic conjunctivitis and corneal disease. Dog breeds prone to developing ectropion include: St. Bernard, Bloodhound, Bull Mastiff, Newfoundland, Great Dane, and the spaniels. Dogs with eyelid damage, hypothyroidism, or facial nerve damage may also develop ectropion. In addition to the exposed inner eyelid, the dog may have tear staining of the face and copious mucopurulent discharge from the affected eye. The cornea may be ulcerated due to exposure or scratching at the irritated eye. Mildly affected dogs can be treated with topical medications such as lubricating eye drops and ophthalmic antibiotics and underlying medical conditions such as hypothyroidism should be treated. Surgical treatment is indicated for more severely affected animals.

A lateral eyelid wedge excision is a surgical procedure performed to correct ectropion in the dog. The surgery consists of removing a full thickness wedge of the lower eyelid. This shortens the length of the lower eyelid and is performed at the lateral canthus for cosmetic reasons. After the wedge is removed, the edges of the incision are closed in two layers. The deeper layer can be closed with 3-0 to 5-0 absorbable suture with buried knots to make sure the cornea is

not irritated by the sutures. The second layer is closed with 4-0 to 5-0 nonabsorbable sutures. Other surgical procedures used to correct ectropion include Kuhnt-Szymanowski procedure, the Kuhnt-Helmbold procedure, a V to Y plasty (Wharton-Jones procedure), and the Munger and Carter modification of the Kuhnt-Helmbold technique.

Cataracts

Cataracts in dogs form from cross-linking of lens proteins. This cross-linking occurs due to any number of factors, including heredity, high blood glucose, low calcium levels, toxins, genetic defects, poor nutrition, synechia formation (adhesion to uveal tissue), or radiation exposure. Most cataracts are an inherited condition due to a recessive autosomal gene. Physical examination reveals an opaque lens. Cataracts in the older animal should not be confused with lenticular sclerosis, which does not cause vision loss. Cataracts obstruct the normal tapetal reflection, appearing as dark spots over the normally bright tapetal reflection. Treatment consists of surgery via ultrasonic lens fragmentation (phacoemulsification); intraocular lenses can be implanted at the same time to correct farsightedness.

Autoimmune Hemolytic Anemia

A dog with autoimmune hemolytic anemia (AIHA) presents with clinical signs of weakness, anorexia, lethargy, respiratory distress, increased heart rate, exercise intolerance, diarrhea, and/or vomiting. Physical examination of a dog with AIHA shows a dog with pale mucous membranes, rapid heart and respiratory rates, heart murmur, icterus, fever, enlarged liver and spleen, and possibly skin hemorrhages. Examination of the blood shows a large number of spherocytes (RBCs that are round, small, and intensely stain without a central pallor), as well as anisocytosis and polychromasia. The dog is also anemic and has leukocytosis with a left shift and a high MCV. A Coomb's test (direct antiglobulin test) is positive in over 60% of dogs with AIHA. Testing of the blood serum reveals increased alanine transaminase (ALT) levels, hemoglobinemia, and hyperbilirubinemia. Urine examination shows blood and bilirubin in the urine.

Atopy

Clinical signs of canine atopy include itching, along with licking and biting. In addition to the allergic condition, complicating diseases may develop in conjunction with atopy. The physical examination reveals skin lesions with redness, crusts, scaling, lick staining, oily skin, lichenification, hyperpigmentation, hair loss, and conjunctivitis. Of note, dogs do not typically sneeze with allergies. Secondary pathogens associated with atopy include yeast (Malassezia), otitis externa yeast infections, or *Staphylococcus* infections. Testing options include skin scrapings to rule out mange/mites, skin cytology to

check for yeast or bacteria, or allergy testing. Allergy testing may involve serum testing for immunoglobulin E (IgE) or intradermal skin testing. Treatment can be any of the following: hyposensitization, antihistamines, or corticosteroids.

Blastomycosis

Treatment of *Blastomyces dermatitidis* is a long-term commitment and is expensive. Amphotericin B is the drug of choice to treat this illness; this is a fairly toxic drug, causing renal toxicity, hypokalemia, anemia, and possibly phlebitis. It can also be used in combination with ketoconazole. The typical dose is .5mg/kg every other day given IV with dextrose. Ketoconazole is another alternative, although given alone, it is not as effective as amphotericin B. It should be given with food as it can cause nausea and vomiting along with anorexia and diarrhea and it can affect the liver. Oral dosing is 10 to 30 mg/kg twice daily for up to three months. A third alternative is itraconazole, which causes side effects similar to those of ketoconazole and should be given with food. The dose is 5 to 10 mg/kg once or twice daily for up to three months. Recovery is guarded in dogs with blastomycosis.

Anal Sac Carcinoma

Anal sac carcinoma is more frequently seen in female dogs older than eight years of age. It is a malignant neoplasm of the apocrine glands of the anal sac and is malignant. Disease invades the surrounding perianal tissues and affects the associated lymph nodes. Laboratory results frequently show a high serum calcium level (paraneoplastic syndrome, which can cause kidney damage) and a low phosphate level. Surgical biopsy gives a definitive diagnosis. The carcinoma presents as a tumor of varying size, possibly greater than 10 cm, and is commonly ulcerated. Biopsy results show carcinoma-type cells and acinar formation. These tumors have a poor prognosis despite removal of primary tumor, hormonal treatment, radiation, and/or chemotherapy.

Aflatoxin Poisoning

Dogs can become poisoned due to ingesting aflatoxin, which is produced by *Aspergillus flavus, Aspergillus parasiticus* , or *Penicillium puberulum* . Dogs that ingest grain or grain-based food contaminated with aflatoxin can display clinical signs. The severity of signs depends upon the amount of aflatoxin consumed and includes loss of appetite, loss of weight, icterus, hemorrhage, ascites, and sudden death. The liver is the primary organ damaged by aflatoxin. Lab tests that may be abnormal in aflatoxin poisoning include increased alanine transaminase (ALT), bilirubin, SAP, blood ammonia, and fibrinogen, decreased albumin, thrombocytopenia, bilirubinuria, and prolonged prothrombin time (PT) and partial thromboplastin time (PTT). Necropsy exams of dogs that have died of aflatoxin poisoning reveal liver abnormalities such as mottled

appearance and fatty changes of the liver, icterus, ascites, and hepatocellular changes.

Brucellosis

Canine brucellosis is caused by *Brucella canis* . This gram-negative intracellular organism causes abortions, epididymitis, testicular atrophy, and reproductive failure. Transmission occurs during breeding or ingestion of the organism through discharges or placentas. The disease can be difficult to diagnosis as some dogs do not show signs. It may be suspected if bitches abort or are infertile. Male dogs with symptoms can have signs such as enlarged scrotums or dermatitis, infertility, epididymitis (enlarged, hardened epididymides), or atrophic testicles. Semen from affected males may be abnormally low or have high levels of immature sperm. Other dogs may have ocular signs, loss of sex drive, lymph node enlargement, spinal pain, or generalized weakness. Diagnosis is based upon blood cultures and serological testing. Treatment options rely upon administration of tetracycline drugs with streptomycin. Gentamicin can be substituted for streptomycin if it is not available. Therapy is not always successful.

Distemper Vaccination

Vaccinations against canine distemper can control the disease and prevent its spread. Vaccination with a modified-live virus vaccine gives a dog long-lasting immunity. The vaccination schedule should first be given at six to eight weeks of age. Maternal antibodies do not interfere with the immunization provided the pup ingested colostrum during the first day of life. Pups that do not get colostrum should not be given the modified-live vaccine until they are a month old, as it can cause fatal encephalitis. Following the first injection, two more vaccinations should be given at three- to four-week intervals in order to prompt an adequate immune response. After the initial three vaccines, boosters should be given yearly to every two years.

Tracheal Collapse

Treatment for a dog with tracheal collapse is aimed at suppressing the cough as much as possible and stopping the progression of degenerative changes in the airways. The first line of treatment after a dog has been diagnosed consists of use of anti-inflammatory drugs to control the cough. Usually, prednisone is used first, starting at a 1 mg per kg dose twice daily tapered to every other day. Occasionally, inhaled glucocorticoids have been used, but this is not a common practice. Some cases require bronchodilators (theophylline) and/or cough suppressants (codeine, hydrocodone, butorphanol) paired with the anti-inflammatory agent. If secondary infection is present, antibiotics should be

used; this is especially important if steroids are given. Occasionally, dogs will benefit from tracheal ring surgery or placement of a tracheal stent.

Exocrine Pancreatic Insufficiency

Treatment of exocrine pancreatic insufficiency is based upon supplementation with dietary enzyme and ensuring that dogs receive proper nutrition. Pancreatic enzymes come in two forms: A powdered form of nonenteric coated preparation to resist the degrading effects of gastric acids is mixed with food (2 teaspoons per 20 kg body weight mixed with each meal). Fresh pancreatic enzymes (3 grams per kg body weight with each meal) can also be given to the dog to supply the enzymes needed to properly digest food. Other nutrients may need to be supplemented, especially the fat-soluble vitamins A and E. It is also recommended to supplement the dog with vitamin B12. A monthly injection of cyanocobalamin (250 to 500 μg subcutaneously) is needed, as use of pancreatic enzymes does not correct the malabsorption of vitamin B12.

Aortic Stenosis

The dog can suffer from three types of aortic stenosis: valvular aortic stenosis, supravalvular aortic stenosis, and subaortic stenosis (subvalvular aortic stenosis). Of these three, the most commonly encountered type is subaortic stenosis. Regardless of type of stenosis, the obstruction causes increased stress on the ventricular wall. This affects left ventricular emptying, leading to left ventricular hypertrophy in an attempt to normalize the ventricular systolic function. Hypertrophy may then create the potential for the myocardium to have areas of ischemia, causing mitral regurgitation and possibly leading to the development of ventricular arrhythmias. Some dog breeds that are predisposed to aortic stenosis are large-breed dogs such as the Newfoundland, Boxer, Rottweiler, German shepherd, and Golden Retriever.

Heartworm

Heartworm disease prevention is mandatory in all climates due to the presence of the infective host, the mosquito, which is a ubiquitous insect. Macrolide drugs such as ivermectin, moxidectin, milbemycin, and selamectin are excellent heartworm preventatives. Ivermectin combined with pyrantel pamoate and milbemycin kills digestive tract nematodes. Selamectin also kills fleas and flea eggs, ticks, ear mites, and mange mites. These drugs work to stop larva development during the first two months of infection. If the preventative treatment is given once monthly starting at two months of age, no testing needs to be done. If the animal is tested after a year of age, an antigen test should be done. Even with year-round preventative treatment, it is recommended that dogs be retested annually to account for any missed doses of heartworm preventative.

Treatment for heartworm disease should start with a thorough evaluation of the dog's cardiovascular system. A serious but common complication of heartworm treatment is the possibility of thromboembolism caused by the dying worms. The adulticide used to treat heartworm is melarsomine dihydrochloride, an organoarsenical. This drug kills both adult and immature worms of either sex. The drug is injected deep into the lumbar musculature in a dose of 2.5 mg per kg in two injections spaced 24 hours apart. In animals with severe cardiopulmonary disease, one dose is given, which kills about 50% of the worms; to avoid thromboembolism formation, the two-injection regimen is initiated in one to three months. Melarsomine dihydrochloride can cause inflammation at the injection site; the dog must be restricted from exercise for one to two months after treatment to lessen the chance of pulmonary embolism.

E. Coli Infection

Escherichia coli , a gram-negative bacterium, is a normal inhabitant of the digestive tract; however, in neonatal puppies, it can cause a life-threatening septicemia. This happens because the pup may have not taken in adequate amounts of colostrum or because of dirty and crowded kennel conditions. Signs in infected puppies include anorexia, vomiting, weakness, diarrhea, hypothermia, or death. This is a life-threatening condition, and proper in-patient treatment is needed to prevent death, although the prognosis for recovery is grim. Fluid therapy, good nursing, and antibiotic treatment need to be implemented. If the bitch is in poor health, the puppies may need to be hand-nursed until the bitch is able to provide adequate amounts of milk.

Accumulation of Copper

Copper can accumulate in the liver of some dogs, causing chronic hepatitis and eventually liver failure. While all dogs can be affected, a few particular breeds are genetically prone to this condition, including the Bedlington terrier, West Highland white terrier, Skye terrier, and Doberman pincher. It is known that the Bedlington terrier transmits this condition via an autosomal recessive gene. Signs can range from acute severe disease and subclinical disease to a chronic progressive disease. Bedlington terriers can experience the acute disease, which manifests as anorexia, vomiting, liver enlargement, jaundice, dehydration, and depression. This breed and the other breeds can also have the more progressive form, with signs that come and go such as not eating, depression, listlessness, vomiting, and diarrhea. Eventually, after many such episodes, the dog may also have liver enlargement, jaundice, a swollen abdomen, and signs of hepatic encephalopathy.

Demodicosis

Demodex canis is a normal inhabitant of the dog's skin. In some dogs, demodex numbers increase to levels that lead to the disease demodicosis. The disease can be localized, causing mild redness and scaling in patches on the face, trunk, and legs or it can be a generalized disease that is widespread and causes severe disease. The disease can occur in young animals that have an immature immune system or in adults. Adults that contract the disease commonly have the severe form as a result of another disease or cancer; this type can be very difficult to treat. Treatment consists of pesticides that kill the mites and bring their numbers under control. Amitraz, ivermectin, and milbemycin regimens are used, but all have side effects that need to be managed.

Acral Lick Lesions

Acral lick lesions are commonly seen on the front side of the legs. They are hairless, thickened, raised, and firm lesions that frequently become ulcerated. Dogs that have allergies, endocrine problems, or other skin issues frequently come down with the lesions. It can also be a behavioral issue or be caused by neoplasia or trauma. The dog chews and licks the affected area, which may become infected with bacteria. Treatment can be frustrating. Any medical causes, such as neoplasia, infections, mites, allergies, or endocrine problems, need to be addressed at the same time treatment of the lesion(s) is attempted. Medications, such as behavior modifying drugs, antibiotics, or anti-inflammatory or anti-allergy drugs, can be used in addition to topical solutions to keep the dog from licking the area.

Hemorrhagic Gastroenteritis

Toy and miniature breeds and puppies are particularly susceptible to canine hemorrhagic gastroenteritis, a disease with no clear-cut cause. It may be caused by endotoxemia of bacteria and/or a type 1 hypersensitivity reaction. *Escherichia coli* and *Clostridium perfringens* have been suggested as the bacteria responsible for this disease. Dogs will present with an acute bloody diarrhea along with loss of appetite, vomiting, lethargy, and possibly a below-normal temperature. Diagnosis is based upon clinical signs, particularly the rapid onset of bloody diarrhea, a PCV greater than 60%, and ruling out other viral, bacterial, or parasitic causes of the diarrhea. Treatment is best initiated with prompt fluid replacement via IV therapy, use of ampicillin and steroids, and transfusion if blood loss is extreme.

DIC

Disseminated intravascular coagulation, otherwise known as DIC, is a very serious disease in the dog in which fibrin thrombi form in the vascular system and coagulation factors are activated. The primary problem is capillaries being obstructed by thrombi and abnormal bleeding. It occurs secondary to severe

diseases such as shock, heartworm disease, systemic infectious diseases, cancer, or snake bite. The disease is diagnosed in very sick animals, which also exhibit some or all of these findings: prolonged prothrombin time and activated partial thromboplastin time, abnormal bleeding (petechiae), high fibrin degradation products, and thrombocytopenia. This disease demands emergency in-patient treatment for both the blood disorders and the main cause of disease. To treat the blood disorders, the dog should be placed on fluids, and after careful consideration, treatment with heparin may be indicated to combat the problems with coagulation. The outcome for an animal with DIC is guarded.

Chocolate Poisoning

Dogs are prone to chocolate poisoning, especially around holidays when chocolate is readily available. Baking chocolate is especially toxic due to the high concentration of the methylxanthines caffeine and theobromine. The lethal dose of caffeine in dogs is 140 mg/kg and that of theobromine is 250 to 500 mg/kg. After ingestion, chocolate is rapidly absorbed. The half-life of theobromine is 17.5 hours and that of caffeine 4.5 hours. Signs of chocolate toxicity include vomiting, diarrhea, hyperactivity, increased urination, ataxia, tremors, increased heart and respiratory rate, seizures, weakness, and/or coma. Death may be caused by respiratory failure or cardiac arrhythmias. Treatment consists of using emetics if no clinical signs have started, such as activated charcoal, and a cathartic. IV fluids should be started and the bladder should be catheterized to prevent the urine from being reabsorbed. Lidocaine (1to 2 mg/kg IV) can be used for arrhythmias of ventricular origin.

Ultrasound Images

Ultrasound is used for imaging the male dog's reproductive organs. The prostate gland surrounds the proximal urethra at the neck of the bladder; it is a bilobed organ with a uniform echogenicity. The urethra runs between the lobes of the prostate and can be either hyperechoic or hypoechoic. The testicle is a smooth, oval-shaped organ that is hypoechoic, with the mediastinum appearing hyperechoic. Ultrasound is used to visualize the female dog's uterus and to determine pregnancy. The uterus appears hypoechoic, and depending on estrus cycle, fluid may or may not be present in the lumen. In using ultrasound to determine pregnancy, at 17 to 20 days after the peak of luteinizing hormone, the chorionic cavity can be visualized as a one- to two-millimeter round vesicle.

Separation Anxiety

Separation anxiety in dogs can be managed through behavior and environment modification and medications. It is a fairly common problem, accounting for up to 29% of behavior problems. The diagnosis of separation anxiety is made when a dog shows anxiety signs when separated from its owner. Signs of

anxiety include vocalization, urination, defecation, and/or destruction of environment. Possible behavioral and environmental modifications include relaxation training, increasing exercise, desensitization to owner departure, and reducing excitement at reunions or departure. Punishment does not work to resolve the problem of separation anxiety in the dog. Medications used to treat separation anxiety include tricyclic antidepressants (the mainstays of pharmacological treatment), benzodiazepines, monoamine oxidase inhibitors, azapirones (buspirone), and antipsychotic agents.

Dominance-Related Aggression

Dogs with dominance-related aggression exhibit the following signs: staring, stiff gait, and carrying the tail and head up. These dogs show aggression towards humans who reach for its food, move the dog from favored sleeping spots (couch or bed), or even people who pet it on the head. The first step in dealing with such an animal is to muzzle the dog and to use physical barriers to prevent humans from being bitten. Behavior modification can be tried to change the aggressive behavior. All attention to the dog should be withdrawn for two weeks while the household determines which behaviors incite aggression. The next step is for the household members to establish dominance over the dog; training should be reinforced (sit, stay, down) and all benefits must be earned (food, walks, attention). This will help control the dog when the situations that previously caused aggression are encountered.

Estrus Cycle Stages

Proestrus : This stage is represented by much activity to prepare the reproductive tract for breeding. The corpus luteum regresses, while follicles grow on the ovaries. The uterus undergoes enlargement due to an increase in glands and thickening of the endometrium. The vaginal cells undergo changes, especially keratinization of the superficial layer, which is important in staging the estrus cycle.

Estrus : During this stage, the female is receptive to the male. The cervix relaxes; there is increase in vaginal mucus. A surge in luteinizing hormone coincides with ovulation, which occurs either during estrus or immediately after estrus.

Metestrus : The corpus luteum forms on the ovary from the remnants of the ovulated follicle. All secretions in the reproductive tract decrease.

Diestrus : This stage is characterized by presence of a corpus luteum and progesterone production. The uterine glands are hypertrophic and the cervix constricts.

Anestrus : The stage of the cycle when there is no reproductive activity.

Vaginal Cytology

Vaginal cytology is frequently used to determine the best time period to breed the bitch. A sterile swab is moistened with sterile water and passed through the sterilized vulva and into the dorsal vagina to obtain a cell sample. The swab is rolled onto a microscope slide and stained using Wright's stain. Epithelial cells, including cornified cells or large basophilic cells with a condensed nucleus, and red blood cells are the primary cell types examined.

Proestrus: Microscopic examination in the early stage will reveal numerous red blood cells and epithelial cells that are primarily non-cornified. Towards the end of proestrus, the cells become more cornified.

Estrus: The smear is very cellular. The red blood cell population decreases and cornified cells are more abundant. These cells will appear as more irregular, with no nucleus and a folded cytoplasm.

Metestrus: After day 2, very few cornified cells are seen on vaginal cytology. If the bitch is not pregnant, non-cornified epithelial cells and neutrophils are seen.

Anestrus: During this stage, squamous epithelial cells with basophilic cytoplasm are primarily seen on vaginal cytology.

Proestrus and Estrus Events

During proestrus, the bitch displays vulvar swelling and a bloody discharge that attracts males. The bitch does not show interest in the male, and due to high testosterone levels, the female may attempt to mount other dogs. Estrus typically lasts nine days, but can be as short as 3 days or as long as 21 days. Estrus is typified by a clear discharge, and the female will display behavior designed to attract males. Both sexes will become interested in each other. The bitch will only allow mating at full estrus, and she will "flag" the tail. Mating commences when the female stands with the tail to one side to allow the male to mount.

Hormones

During early proestrus, estrogen levels are low but continue to rise and reach their peak near the end of this stage. Progesterone levels then begin to rise, and estrogen levels decrease. Progesterone remains high during the next two stages and can be measured to indicate when a bitch will ovulate and should be bred. At the start of estrus, there is a short surge in luteinizing hormone (LH). At the peak of the LH surge, ovulation occurs

between 24 to 48 hours. The best time to breed the dog is four days after the LH surge. Progesterone levels are high during this stage. Progesterone is the main hormone present during diestrus. During anestrus, hormone levels are low, although near the end, estrogen levels begin to rise.

Birds

Suspected Feather Picking

Diagnostic tests should be performed in cases where feather picking is suspected in a pet bird. A basic diagnostic database includes a chemistry panel and complete blood count. The cloaca, mouth, and nares should be swabbed and a gram stain, bacterial culture, and fungal cultures should be done on the swabs. Feather and skin scraping may be warranted as well. Other testing should be done if the physical exam and history suggest that a certain medical condition might be the cause of feather picking. Treatment is dictated by medical tests results. If the bird has self-mutilation syndrome, an Elizabethan collar can be placed on the bird for a week or two to break the cycle. If it is a behavioral problem, the inciting cause(s) must be identified and corrected. Some cases have been treated by medications, such as antidepressants, antianxiety medication, steroids, and hormones, and/or referred to bird behavioral specialists.

Psittacine Beak and Feather Disease

Psittacine beak and feather disease is a fatal disease caused by a small DNA circovirus. It is a progressive disease that is thought to be specific to psittacines, and all the psittacine species are susceptible, especially Cockatoos, Lovebirds, Macaws, and African gray parrots. Young birds are more likely to die from the disease than older birds, which may become carriers for the virus. The virus is transmitted to birds from inhalation or ingestion of the virus, from direct contact with infected birds, indirectly from a contaminated environment or equipment, and from crop feeding, feather dust, and feces. Signs include loss of feathers, abnormal new feathers, abnormal beak, nail lesions, and loss of powder down. Diagnosis is made by electron microscopy, histopathology, or PCR testing.

Tassle Foot

Scaly leg and face mites are mites of the genus*Knemidokoptes* that act as parasites of the beak and skin of birds. The condition is named scaly leg and scaly face or tassle foot because the mites can cause tassel-like growth on the feet of smaller pet birds. Other signs of infestation include scaly white or gray lesions on the skin that does not have feathers. Canaries commonly exhibit

tassel-like growths on the feet; other lesions are found on the beak, the eyes, and the vent. In some cases, the feet, toes, and beak are malformed. The treatment for scaly leg and face is ivermectin. Two or three ivermectin treatments at 10-day intervals may be needed. The drug can be applied topically, given via injection, or given orally. All equipment used by the bird should be cleaned and disinfected while treatment is being given.

Deficiency of Vitamin A

Hypovitaminosis A is a deficiency of vitamin A in pet birds. In pet birds with this condition, epithelial cells are the tissues affected. Squamous epithelium (skin or mouth) becomes thickened by excessive keratin production. Other epithelial cells such as those found in the kidney or upper respiratory tract can be affected by excess keratin production as well. Early in the disease course, the bird's eyes can become dry and the third eyelids are thickened. The bird may present with sneezing, sinusitis, scaly skin, and poor feather quality. As the disease progresses, sterile abscesses form in the mouth glands, the bird experiences respiratory distress, and renal gout may occur. Treatment includes supplementing vitamin A in the diet and/or giving vitamin A injections.

Psittacosis

Psittacosis (avian chlamydiosis) can affect birds and mammals. Psittacine birds are particularly prone to developing the disease and are important sources of infection in humans. The infection in humans causes respiratory disease. They can become quite ill with flu-like symptoms. Serious complications can occur, such as pneumonia, failure of organs, and even death. Birds with the disease may be healthy-appearing carriers or may have an acute or chronic illness. The organism that causes the disease is *Chlamydophila psittaci* , an obligate intracellular bacterium. It is passed from the infected bird in the feces or in discharges from the respiratory tract in elementary bodies. Tetracyclines are effective against *C. psittaci* as are most common disinfectants and heat.

Egg Binding

Egg binding is a common condition affecting pet birds. Lovebirds, parakeets, and cockatiels are prone to develop this life-threatening condition. The bird that becomes egg-bound has a history of egg laying. The bird may have anorexia, refuse to perch, and act depressed. The first treatment option is to treat the bird medically. Drugs used include oxytocin and prostaglandins to help the uterus contract and to help the egg descend. Birds that exhibit signs of shock should be given a dose of steroids and antibiotics as well. If the egg does not pass, the bird should be anesthetized and the egg manually extracted. Steady pressure by the fingers on the upper abdomen will cause the egg to

drop. Complications include the egg being stuck in edematous tissues, as well as septicemia and uterine rupture.

Erysipelas

Erysipelas is caused by the intracellular bacterium *Erysipelothrix rhusiopathiae* , which is a facultative anaerobe. It causes outbreaks of acute septicemia in flocks, and mortality may reach up to 50% of the flock. Turkeys are the species most frequently afflicted, although other domestic poultry flocks, pigs, and reptiles have also been infected. Erysipelas is also a zoonotic disease. The disease in humans is called erysipeloid and infection may occur through skin breaks. In humans, the disease can either remain localized or it can spread throughout the body and cause death. Treatment of infected birds can be done by adding penicillin or broad-spectrum antibiotics to the drinking water. Turkeys can be vaccinated to control the disease in turkey flocks.

Fowl Cholera

Pasteurella multocida causes fowl cholera, a highly contagious disease of all birds; it is spread through infected bird excretions that contaminate the environment. The disease causes an acute form in which birds suddenly die. Other affected birds show clinical signs such as rapid breathing, diarrhea, anorexia, fever, oral discharges, and unkempt feathers. The disease may also take on a chronic form. Swellings can occur in the joints, breast bursa, and footpads. Discharges from the eye and oral cavity indicate infection in these areas. Sometimes CNS signs are seen if the infection sets in the middle ear or meninges. Treatment based on antibiotic sensitivity selection should be employed. Tetracycline, penicillin, or sulfas placed in the water or feed are usually effective.

Bovine

Reproductive Cycle

Twin pregnancies occur in up to five percent of cattle; the rate increases as the age of the dam increases. A female twin with a male twin has a good chance of becoming a freemartin due to the effect of male hormones such as testosterone on the female. The bovine has 60 chromosomes; the cow's estrus cycle is 21 days and pregnancy lasts about 280 days. After the egg is fertilized, it reaches the two-cell stage at 48 hours, the four-cell stage at 56 hours, the eight-cell stage at 73 hours, and the morula (16-32 cells) after 96 hours. Implantation occurs 20 days after fertilization. The placenta is a polycotyledonary placenta and weighs about 4 kg. The cotyledons are on the placenta, while the caruncle is on

the uterus. Together, the cotyledon and maternal caruncle are called the placentome.

Rumen Microbial Population

In the ruminant, digestion begins in the rumen. Microbes in the rumen ferment or modify the food ingested, and then the ruminant absorbs these products. This microbial action allows the ruminant to extract energy from fibrous plant material that monogastrics cannot utilize. Proteins are a byproduct of this microbial action, and this is the source of most of the ruminant's protein needs. The microbial population in the rumen is a dynamic population but generally consists of bacteria, small amounts of anaerobic fungi, and ciliated protozoa. The main products produced by these microbes are volatile fatty acids and microbial protein. The volatile fatty acids are the main energy source for the ruminant and microbial protein is the main source of protein for the ruminant.

The rumen microbial population consists of bacteria, protozoa, and a small number of anaerobic fungi. The rumen is a strictly anaerobic environment. The composition and number of the population can change quickly due to diet, illness, or feeding changes. In a "typical" rumen, there can be 1010 bacteria/mL, 106 protozoa/mL, and 103 fungi/mL. Bacterial numbers are low right after feeding, but gradually increase until 16 hours after feeding. There are two types of protozoa in the rumen: Holotricha protozoa help to control the rate of carbohydrate fermentation when large numbers of soluble sugars are included in the diet; the entodiniomorph protozoa digest starch granules to control the rate of starch digestion.

Intravenous Injections

Intravenous injections in cattle are given in the external jugular vein, the caudal tail vein, and the subcutaneous abdominal vein. Rarely are injections given in the auricular or recurrent tarsal veins. The most common site on the bovine to give an injection is in the external jugular vein after the animal has been restrained and the head is turned to the side to expose the neck. A two- to three-inch, 14- to 18-gauge needle is commonly used. The caudal tail vein can be used to give small doses of medications. The subcutaneous abdominal vein is very delicate and frequently subject to hematoma formation. A smaller gauge needle should be used than that used to give jugular injections.

Inhalation Anesthesia

There are many general considerations involved in the use of inhalation anesthesia in large animals. Proper equipment and an adequate number of personnel should be available when using inhalation anesthesia. The animal

should be intubated to maintain an airway and to prevent aspiration of stomach contents into the lungs. Bloat can quickly occur in a recumbent animal, and due to the weight of the animal, radial paralysis is a concern. To prevent aspiration and bloat, food and water should be withheld prior to surgery. The down side of the animal should be adequately padded and the shoulder should be placed in extension. Surgery should be completed thoroughly but quickly to minimize time spent in a recumbent position.

Bull Breeding Soundness Exam

A bull breeding soundness exam consists of three steps: A physical examination, both general and specific for reproductive organs, should be performed; the scrotal circumference is then measured. The final step is to collect a semen sample for evaluation. Based upon these examinations and measurements, bulls are categorized into three types: satisfactory potential breeders have met all minimum values and are free from genetic, skeletal, or infectious defects; unsatisfactory potential breeders fail to meet minimum values and have defects that impact breeding potential. The third type, classification deferred, indicates that the bull cannot be excluded from the satisfactory category as it may improve given time or with appropriate therapy; later retesting will place the bull in either the satisfactory or unsatisfactory category.

Examination of sperm motility and morphology is an important component of a bull breeding soundness exam. Motility of 30% is considered the minimum amount to rate a bull as satisfactory. Rapid swirling of sperm is considered very good, slower swirling good, generalized oscillation fair (minimum threshold), and sporadic oscillation poor. Sperm morphology should have a minimum of 70% normal cells. Abnormalities can be primary: underdeveloped, acrosome defect, doubles, small or free abnormal heads, abnormal midpieces, crater defects, pear-shaped defects, proximal droplet, strongly coiled or folded tails, narrow heads, or accessory heads. Secondary abnormalities include: free normal heads, small normal heads, abaxial implantation, distal droplets, simple bent tail, giant or short broad heads, loose acrosomal membranes, distal droplets, simple bent tails, or terminally coiled tails. Other cells may be seen, such as red blood cells (RBCs), white blood cells (WBCs), epithelial cells, sperm precursor cells, round cells, and cells in medusa formation.

Pregnancy Diagnosis

Pregnancy diagnosis is an extremely important part of cattle production. It is to the producer's economic advantage to have accurate diagnosis early in the pregnancy or soon after breeding so that non-pregnant female bovines can be rebred. It is also helpful to learn an expected due date. Dairy cattle are

generally palpated for pregnancy 28 to 35 days after being bred. At 28 days, a corpus lutea is present on the ovary on the side of the pregnant horn; the horn is also enlarged and fluid-filled. The four positive signs of pregnancy are an amniotic vesicle present at 30 to 70 days, chorioallantoic membrane (membrane slip) at 30 to 35 days, placentomes at 65 to 70 days, and the fetus, which can be felt at 65 to 70 days. Beef cattle are generally palpated at a later stage of pregnancy.

Bowel Intussusceptions

Cattle can develop an intussusception of the bowel, which is telescoping of a piece of bowel into an adjacent segment. This should be differentiated from neoplasia and abomasal ulcers. Although it does not occur with much frequency, in adults, the jejunum is a likely site. In calves, intussusception can be associated with enteritis. Clinical signs include anorexia, depression, colic, abdominal distention (because the rumen fills due to obstruction), decrease in milk production, and decrease in manure. As the condition progresses, the animal becomes dehydrated, passes dark, reddish stool, and develops shock. Treatment includes surgical resection of the damaged intestine, fluid and electrolyte replacement, antibiotics, and analgesia. Treatment is favorable if the condition is recognized early in development and if peritonitis does not develop.

Minimizing Pneumonia

One important component of managing pneumonia in feedlot cattle is to purchase preconditioned calves. This means the calves have been castrated and dehorned, vaccinated against respiratory diseases, treated for internal and external parasites, and weaned and on feed at least three weeks before moving off the farm of origin. When the calves arrive at the feedlot, they should be processed within two days of arrival by a vaccine booster, treated for parasites, implanted, and given mineral and vitamin supplementation if deemed necessary. The calves should be closely monitored by trained personnel at least twice daily for one to two weeks upon arrival. Group treatment with antibiotics should only been done upon arrival if a group shows signs of unkempt appearance, coughing, or other respiratory signs, or if calves were not preconditioned, had a long transportation distance, or were bought at auction houses.

Traumatic Pharyngeal Injury

Treatment of traumatic pharyngeal injury includes drainage of abscesses into the pharynx. Chemical restraint should be avoided in order to prevent aspiration of drained material into the lungs. The animal should be securely restrained while the abscess is punctured. Once material has drained from the

abscess, the wound should be flushed with an iodine-based solution. In addition to lancing, antibiotics, such as sulfas, tetracyclines, ampicillin, or procaine penicillin, should be given for seven to 14 days depending on extent of infection. Anti-inflammatory agents such as aspirin, *flunixin, or meglumine,* relieve swelling and give pain relief. Water access should be unrestricted; a stomach tube may be used if the animal refuses to drink. Soft mash or soft grass should be offered as well.

Calf Diphtheria

Necrotic laryngitis or calf diphtheria is a common acute or chronic laryngeal infection of calves from three to 18 months of age. The causative agent is *Fusobacterium necrophorum* , which gains access to the laryngeal mucosa through open sores or ulcers, frequently caused by upper respiratory infections. The disease produces clinical signs of a moist, painful cough, nasal discharge, salivation, fetid breath, necrotic ulcers on the arytenoid vocal processes, and a loud inspiratory dyspnea. An affected calf frequently stands near the water source, taking frequent small sips of water. Along with these signs, a high fever, anorexia, and reddened mucous membranes may occur. Treatment should be aggressive, using intramuscular broad-spectrum drugs, such as sulfonamides, procaine penicillin, and Tilmicosin,) along with nonsteroidal anti-inflammatory drugs (NSAIDs), such as aspirin, flunixin, and meglumine, and good nursing care.

Bronchopneumonia

Destruction of the pathogen is done with judicious use of appropriate antibiotics. The first choice should be antibiotics labeled for use in food animals and withdrawal times should be strictly followed. Ideally, treatment should be based upon culture and sensitivity. In lieu of this, antibiotics should be given based upon the most likely causative agent, likelihood of client compliance to follow through with treatment, and severity of disease. IV administration achieves high plasma concentrations that penetrate deep into affected lung tissue. The body temperature of the animal should be taken daily. If no improvement is seen within two days, the antibiotic choice should be reconsidered. In addition to antibiotics, anti-inflammatory agents, primarily NSAIDs, should be given and an antihistamine should be considered as well. Good nursing care, including fresh water, adequate feed and shelter, and a clean environment, greatly enhances treatment.

Dictyocaulus Viviparous

Verminous bronchitis and pneumonia in cattle are caused by one of two parasites: *Dictyocaulus viviparous* and migration of *Ascaris suum* larva. With

Dictyocaulus viviparous , yearling cattle on infected pastures are affected more often than adult cattle. The cattle ingest the infective third-stage larva with the grass; the larvae then penetrate the intestine and migrate to the mesenteric nodes. In the nodes, the larvae molt into fourth-stage larva and pass into the lymph and blood circulation to eventually lodge in the lung capillaries. One week after ingestion, they enter the alveoli, molt, and set up in the bronchi. Adults are present in the lung three to four weeks after ingestion. The adults then begin to hatch into eggs, which are coughed up, swallowed, and passed out of the body in the feces.

Lung worm, or *Dictyocaulus viviparous* , is more common in yearling cattle on the pasture and is more common in late summer and fall or after periods of high rainfall or irrigation. Clinical signs of *Dictyocaulus viviparous* infection include coughing, increased respiratory rate, fever, loss of appetite, and loss of weight. Depending on severity of infestation, which correlates with the number of larva present in the bronchi and lungs, pneumonia may be present. Upon auscultation, the examiner hears crackles, wheezes, and harsh breath sounds. The caudal lung lobes become consolidated and subcutaneous emphysema may occur. More severe cases may even lead to death. Diagnosis is made through clinical signs and demonstration of eggs in the feces using the Baerman sedimentation technique. Transtracheal wash may reveal an increase in eosinophils. Treatment involves use of anthelmintics, especially ivermectin.

Actinobacillosis

Actinobacillosis, or wooden tongue, is caused by *Actinobacillus lignieresii,* a normal inhabitant of the oral cavity. Injury to the soft tissues around the head causes the organism to form granulomatous abscesses, very commonly in the tongue. *A. lignieresii* is a gram-negative saprophyte and is a sporadic problem unless a herd is fed exceptionally coarse feed. Clinical signs of actinobacillosis include a hard and swollen tongue that may protrude from the mouth, stomatitis, painful prehension of food leading to dropping food from the mouth, stridor, an enlarged and firm submandibular area, dehydration, and weight loss. Diagnosis is based upon clinical signs, direct smear of exudates (gram-negative rods and sulphur granules), and involvement of the soft tissues only. Treatment consists of IV sodium iodide, penicillin or tetracycline, and soft food.

Actinomycosis

Actinomycosis, frequently referred to as lumpy jaw, is caused by *Actinomyces bovis* , a gram-positive, branching filamentous organism that is a normal inhabitant of the mouth. It is a fairly common disease of cattle and causes osteomyelitis of the jaw, mostly in the mandible. The organism enters the jaw

through teeth eruptions, oral abrasions, and punctures due to dental disease or feeding on coarse stems or plant awns. It causes hard, nonpainful swelling in the bone; this swelling can rupture and drain fluid that contaminates the environment. An oral examination is necessary to rule out similar-appearing diseases such as tooth root abscesses, tumors, or fractures. Diagnosis is based upon history of trauma, physical examination, and culture of the exudates. Treatment with iodides, penicillin or streptomycin, isoniazid, or flushing of fistulous tracts with povidone iodine can be attempted but commonly does not reduce the size of the mass.

Viral Diarrhea

Bovine virus diarrhea is caused by the bovine virus diarrhea virus (BVDV), a Pestivirus that is a single-stranded RNA virus. It is most common in young cattle ages six to 24 months. There are cytopathic (CP) or noncytopathic (NCP) biotypes: the CP biotype is responsible for the mucosal disease while the NCP biotype is responsible for transplacental infection causing abortions, weak calves, and persistently infected (PI) calves. PI cattle that contract both the NCP and CP biotypes die. Biosecurity measures prevent introduction in closed production systems. The virus is unstable at extremes of pH, and heat kills the virus. It is susceptible to disinfectants and is not viable in the environment after two weeks. Vaccines, both inactivated and modified live, are available. Manufacturers formulate the vaccine to account for viral biotypes and both genotypes 1 and 2.

Healthy Rumen

Normal values found in a healthy rumen environment consist of pH 6 to 7, VFA 60 to 120, and acetic acid levels greater than propionic and propionic greater than butyric acid levels; this is achieved when the bovine is fed a high-quality, long fiber length forage diet. The protein levels are moderate, the crude fiber is greater than 18%, and concentrate supplement is no more than 50% of the diet. Problems arise when producers adjust the diet to achieve greater milk or meat production; thus, new diets need to be carefully adjusted and gradually introduced to give the rumen microflora a chance to adjust. For example, a high-concentrate diet (greater than 50%) with less forage, either in percentage or in length, decreases the rumen pH, leading to a rumen acidosis. While this type of diet does enhance meat and milk production, the acidosis problem can lead to laminitis, ketosis, and parakeratosis.

Grain Overload

Differential diagnoses to consider when dealing with a bovine with suspected grain overload include poli encephalomalacia, peritonitis, coliform mastitis,

parturient paresis, and urolithiasis. Treatment for simple cases of grain overload consists of restricting the affected cattle from grain and water for 18 to 24 hours, while still allowing some access to hay. In addition, giving an antacid such as magnesium hydroxide along with rumen probiotics can be of some benefit. Other treatment options include activated charcoal and mineral oil. More severe cases are considered emergencies. Slaughter may be the best option. Otherwise, IV fluid therapy, emptying the rumen through a rumenotomy, water restriction for 24 hours, and a transfaunation may benefit the animal. If the animal survives, allow access to hay when recovering. Lactic acid has a corrosive effect on the rumen, and secondary sequela such as a mycotic infection, abscesses, or peritonitis can occur despite treatment.

Function of Esophageal Groove

When a calf is born, it has the same stomach arrangement as an adult bovine; however, the abomasum functions much like a monogastric animal's stomach, while the other three compartments are small and essentially do not function during the first few days of life. The reticulorumen takes up to four months to start functioning on an adult level and may take up to a year to fully develop to adult size. When a neonate calf nurses, liquids bypass the reticulorumen via the esophageal groove. Nursing, or rather the proteins and salts in the milk stimulate the glossopharyngeal nerve reflex, forming the groove. This groove, or tube, is formed by two "lips" that course from the cardia to the reticuloomasal orifice, shunting the milk directly to the abomasum. As the calf gets older, this reflex lessens, especially by the time the calf is four months old.

Rumen Development

The calf is not born with a functioning reticulorumen. Diet strongly influences rumen development in the young bovine. Intake of dry feed stimulates the nerves/reflexes that make the rumen function and also inoculates the rumen with the bacteria and protozoa necessary for digestion of food. Calves as young as one week old can begin to eat feeds to initiate this process. Large amounts of feed distend the rumen, which in turn, stimulates the rumen motility and begins to increase the overall size of the rumen. Dry feed should not just consist of concentrates. Some forages, either in hay or in pellet form, should be given to prevent the calf from acquiring chronic rumen acidosis or parakeratosis. Volatile fatty acids stimulate mucosal development, an important component in overall digestion.

Vagal Indigestion

Clinical signs of vagal indigestion include anorexia, scant manure, indigestion, "papple" shape to the abdomen (pear-shaped on the right side, apple-shaped on

the left side), dehydration, and loss of weight. Diagnosis is based upon physical examination, clinical signs, and findings on exploratory laparotomy. Treatment consists of an exploratory laparotomy to determine the cause of the condition. In cases of abomasal volvulus or displaced abomasums, the condition can be corrected through the laparotomy. Induction of birth may be necessary if the fetus is causing the vagal indigestion. Other causes, especially ones involving infection or abscesses, may be more difficult to treat. A rumenotomy may be necessary to remove accumulated ingesta and fluid. IV fluids and a transfaunation may be of some value, along with antibiotic and analgesia use. Prognosis in these cases is not good.

Ketosis

Signs of ketosis can be subclinical or overt. The bovine is usually selectively anorexic, refusing concentrates but still consuming hay. Drastic amounts of weight are lost as body fat stores are mobilized for use as an energy source. Milk production decreases and the cow may have signs of colic. Manure production may also cease. More severely affected cattle begin to show CNS signs as the brain is starved of glucose; the animal may bellow, head press, constantly lick surfaces, appear to be blind, stagger, or exhibit hyperesthesia. Some people may detect an acetone odor to the cow's breath. Lab testing shows increased ketones in the blood and urine and hypoglycemia. Treatment consists of IV glucose administration, glucocorticosteroid, and oral administration of propylene glycol.

Liver Abscess Formation

Liver abscesses are more frequently found in beef cattle than in dairy cattle because beef cattle are typically pushed with concentrates, which causes lactic acidosis rumenitis. This is an important economical consideration at slaughter, as livers with abscesses are condemned for human food use. The organism usually found in liver abscesses is *Fusobacterium necrophorum* , a gram-negative anaerobe, which is a normal inhabitant of the rumen. Causes include rumenitis, naval infection in young cattle, hardware disease, ulcers of the rumen, and bacterial emboli from the portal vein to the liver. Most cases are usually subclinical and found at slaughter. Occasionally, an abscess can rupture and lead to peritonitis, septicemia, or caudal vena caval thrombosis. Prevention consists of slowly adapting cattle to high-concentrate diets and using chlortetracycline in the diet.

Traumatic Reticuloperitonitis

Diagnosis of traumatic reticuloperitonitis, or hardware disease, is based upon physical examination, clinical signs, and laboratory testing. Cases are diagnosed

by clinical signs, such as pain, anorexia, and decreased milk production, lab work indicating infection, abdominocentesis, and laparotomy. Chronic cases may be more difficult to diagnosis. Pericardial complications may lead to distended jugular and abdominal veins. Differential diagnoses include liver abscesses, abomasal ulcers, indigestion, lymphosarcoma, forelimb laminitis, surgical infection, pyelonephritis, fat necrosis, diaphragmatic hernia, or rupture of abscesses. Treatment is based on conservative medical therapy, such as giving the cow a magnet, broad-spectrum antibiotics, and analgesia. If medical therapy does not relieve symptoms, a rumenotomy may be considered to remove the metal object without disturbing any adhesions.

Peritonitis

Peritonitis is an inflammation of the mesothelial lining of the abdominal cavity and the lining that covers the viscera. Because cattle are very good at "walling" off infections, many cases of peritonitis are localized; however, it can become diffuse and present as an acute case or as a chronic case. Clinical signs are quite variable but can include anorexia, depression, and decrease in milk production. Acute cases are typically painful, with the bovine arching its back and also displaying an increased heart rate and fever. Chronic cases have abdominal pain along with signs of shock, such as increased temperature, heart rate, and respiration, along with increased capillary refill time and injected mucous membranes. Generally, digestive tract activity ceases. Diagnosis depends on clinical signs, history, paracentesis of abdominal fluid, and possibly an exploratory laparotomy. Treatment includes excellent nursing and nutrition, broad-spectrum antibiotics, with culture and sensitivity testing of abdominal fluid, fluid therapy, steroids, and nonsteroidal anti-inflammatory drugs (NSAIDs). Localized peritonitis has a more favorable outcome than diffuse peritonitis.

Johne's Disease

Johne's disease is a costly disease caused by *Mycobacterium paratuberculosis* . Most frequently, young adult cattle show clinical signs of Johne's disease, although many cattle in affected herds have subclinical disease. *M. paratuberculosis* causes granulomatous enteritis of the ileum, cecum, and related lymph nodes. This change in the intestine causes a malabsorption of protein (protein losing enteropathy) and diarrhea. The transmission of *M. paratuberculosis* is through the fecal-oral route, but transmission can also be intrauterine or through the milk. Signs of Johne's disease can mimic other diarrheal diseases or diseases that cause weight loss. If a carrier shows signs, these signs can include diarrhea, muscle wasting due to protein loss, weight loss, anorexia, dehydration, and eventually death.

Milk Fever Prevention

Prevention of hypocalcemia (milk fever or postparturient paresis) in the cow begins during the dry period with appropriate feeding. The goal is for the cow to be able to mobilize calcium when it begins to lactate to provide enough calcium in the bloodstream for proper muscle and cardiac function. Low calcium levels during the dry period stimulate levels of parathyroid hormone to remain high. Increasing vitamin D levels in feed five to seven days before the due date also reduces the incidence of milk fever. Other strategies include an IV dose of vitamin D eight days prior to calving and giving high levels of calcium in the feed the day before, during, and the day after calving.

General Anesthesia

Surgery in cattle is usually done under regional anesthesia; if they require general anesthesia, they should be fasted before surgery. Adult cattle should fast for 18 to 24 hours and be taken off of water for 12 to 18 hours. Calves should fast for 12 to 18 hours and be taken off of water for eight to 12 hours. These precautions are needed to prevent aspiration pneumonia, tympany, or regurgitation. Sedation in cattle is usually achieved through use of alpha-2 agonist drugs, such as xylazine, romifidine, medetomidine, or detomidine; xylazine is most commonly used in the US, and at higher doses, can be used for restraint. Some breeds such as Herefords and Brahmans are more sensitive to this drug. Xylazine causes hyperglycemia, hypoxemia, hypoinsulinemia, and hypercarbia. It can also cause pulmonary edema and have an oxytocin-like effect on the uterus.

Urolithiasis Surgery

There are three types of surgery that can be performed on a steer with urolithiasis. In cases where the urethra or bladder has not ruptured, stone removal surgery can be performed. The incision is made cranial to the scrotum and the urethra is opened. The stone is massaged out or crushed and removed. After removal and flushing, the wound is left open to heal by second intention. If the urethra is ruptured, a penile amputation is performed. The skin along the abdomen is incised to let the urine drain before the penis is amputated. If the bladder has ruptured, an ischial urethrotomy is performed. A catheter is placed for five days after the urethrotomy, and both the bladder and surgical wound are left open to heal by second intention.

Lead Poisoning

Lead poisoning in cattle occurs when cattle, with their indiscriminate eating patterns, lick or chew on lead objects such as batteries or drink used motor oil

contaminated with lead. After a period of time of ingesting lead, the disease manifests as encephalopathy. After ingestion, the lead is deposited in the bone where it disrupts heme synthesis, causing shortened red blood cell life and basophilic stippling. From here, it can enter the brain, causing hemorrhage and edema. The signs of lead poisoning include ataxia, excitement, bellowing, blindness, head pressing, and possibly death. Some animals have seizures, diarrhea, or bloat. Treatment involves administering chelation therapy with calcium disodium EDTA, supportive care to ensure proper nutrient and water intake, and removal of the lead from the digestive tract.

Calf Meningitis

A calf with meningitis is difficult to treat. Treatment should begin as soon as possible and be aggressive. Despite treatment measures, many calves still die if afflicted with meningitis. Ideally, antibiotics are chosen based upon culture and sensitivity, but the results are not always available immediately. The best antibiotics are those that kill the bacteria. Compounding the problem is the fact that some antibiotics do not cross the blood-brain barrier despite having high concentrations in the bloodstream. Antibiotics that penetrate the blood-brain barrier include ceftiofur, cefotaxime, doxycycline, erythromycin, isoniazid, minocycline, moxalactam, pyrimethamine, trimethoprim/sulfonamide, and the sulfonamides. The best way to administer an antibiotic is by the intravenous route (if it is available in that form); the antibiotic should be continued for 10 to 14 days.

Urinary Tract Infections

After a bovine has been diagnosed with a urinary tract infection, the urine should be cultured following midstream urine collection. The urine can be gram-stained, and culture and sensitivity testing can direct the use of antibiotics. One of the primary causative agents is *Corynebacterium renale* , which is sensitive to procaine penicillin G (dose at 22,000 to 44,000 IU per kg intramuscularly twice a day), or ampicillin (dose at 11 mg per kg intramuscularly twice a day). Treatment should be started early and aggressively maintained for two to three weeks. If *Escherichia coli* is the responsible organism, ampicillin, trimethoprim-sulfadiazine, or ceftiofur should be used because the organism is resistant to penicillin. The animal should be offered salt to encourage water intake and clean, fresh water should be available at all times.

Leptospirosis

Leptospirosis is a spirochete that infects many organ systems in the bovine including the kidney and reproductive system. While there are 180 serovars, *L*

hardjo is host-adapted to the bovine and is considered to be the reservoir for this serovar. The organism resides in the kidney and/or genital tract but rarely causes kidney dysfunction, and the animal sheds the organism, contaminating the environment and spreading it to other animals. In some cases *L hardjo* also causes a condition known as flabby udder mastitis. It will also cause infertility, stillbirths, weak calves, and abortions. If a bovine is infected with other serovars, primarily *L pomona* or *L grippotyphosa* , severe hemolytic disease and kidney disease can occur.

Listeria

Cattle infected with *Listeria monocytogenes* can shed the bacteria in the feces if they are asymptomatic carriers. The bacterium is hardy, surviving for long periods of time in the environment, up to two years in dry soil. If silage is properly ensiled, the organism can only live for one to two weeks. If a bovine does have clinical signs due to an infection with *L. monocytogenes* , treatment should begin as soon as signs occur. Treatment with tetracyclines or penicillins at high doses is the most effective. Taking the animal off of silage and providing isolation for up to a month help stop the introduction of new infection to herd mates. IV treatment to correct electrolyte imbalances may be necessary. Due to the neurological signs, the animal's pen should be slip-free and well bedded.

Dower Cow Syndrome

Clinical examination of a downer cow should be performed to diagnose the cause of recumbency. The inciting cause, such as electrolyte imbalance or infection, should be corrected before attempting to force the animal to rise. If the animal is bright and alert and responds to immediate treatment, it can be stimulated to stand. If it attempts to rise, assist it by grasping the tail and helping it to stand. Some cases may require goading or the use of lift devices. If the cow is unable to rise, good nursing care is needed. The pen should be deeply bedded and good footing must be ensured. The animal should be rolled from side to side every three hours. Food and water should remain within reach of the animal. It may take up to two weeks for recovery; however, those affected by lymphosarcoma, coxofemoral luxation, or limb fractures will not recover.

Bovine Leukosis Virus

Bovine leukosis, also known as lymphosarcoma, leukemia, or malignant lymphoma, is caused by the bovine leukosis virus. This virus is spread by the transfer of infected blood lymphocytes through shared needles, trauma, surgery, or through insects that draw blood. The virus can also be transmitted to the fetus from infected cows, but this is a rare way of transmission. Colostrum and milk transmission is extremely rare. Most cattle infected with the virus do not

display any signs. The virus is detected through serological testing for antibodies against the virus, such as the agar gel immunodiffusion test or ELISA. About one third of infected cattle have persistent lymphocytosis but no clinical signs.

There are four syndromes associated with bovine leukosis virus infection. The most common form is the adult form, enzootic bovine lymphosarcoma. Cattle with this form have tumors on the right atrium, in the spinal cord, in lymph nodes, or in various other body organs. The signs observed depend upon the organ affected; spinal lymphosarcoma causes paralysis. The other three forms are very rare. Calves can contract juvenile sporadic lymphosarcoma in which there is widespread organ involvement. Older calves exhibit a thymic form that causes a greatly enlarged thymus. The final form is skin leukosis, which causes raised nodules with ulcerated centers. Sometimes these nodules regress but may recur as generalized lymphosarcoma. These three final forms are considered sporadic, as there is no evidence that they are contagious.

Milking Machines

Many factors cause the spread of mastitis in the dairy cow herd. Because the following factors associated with machine function have been found to contribute to mastitis, milking machines should be maintained: vacuum fluctuation at the teat end, high vacuum level, blockage of air admission holes, wide-bore liners, short liner length, and pulsation rate abnormalities. Milking procedures that should be implemented to control the spread of mastitis-causing organisms include wearing of rubber gloves by milkers, stripping teats to check for mastitis, wiping debris from teats, use of pre-dips, wiping pre-dips after adequate contact time, applying the milking unit without admitting air and shutting off the vacuum before removal, and using teat dip after milk out.

Grass Tetany

Grass tetany (hypomagnesemia) is a disorder caused by low magnesium ion concentrations in the blood and cerebrospinal fluid. It is frequently accompanied by low blood calcium levels. The disorder only affects ruminants and causes high levels of mortality. Beef cattle that have recently calved and have been placed on cool season pasture grasses are particularly susceptible to grass tetany. Cattle affected by grass tetany isolate themselves from the herd and have anorexia. They may become aggressive and are hypervigilant, with erect ears, twitching, and hyperesthesia. As the condition progresses, these cattle have muscle tremors, bellow, stagger, and eventually collapse. They also have nystagmus and snapping eyelid retraction. Body temperatures rise due to increased motor activity and respiratory and heart rates increase. Death occurs

during seizure activity. Treatment is through use of commercial IV solutions containing magnesium, typically coupled with calcium.

Prevention of grass tetany or hypomagnesemia should be implemented in herds that have pastures with magnesium-deficient grasses. Pasture grasses or forages with a high magnesium cultivar can be planted if they are available. Susceptible herds can be supplemented with magnesium or the soil can be supplemented with magnesium. Magnesium supplementation should be done on a daily basis by use of mineral blocks or licks, water treatment, spraying magnesium salts directly onto pasture, or by treating each cow with a sustained release magnesium bolus. One ounce per head per day will be sufficient for herds with moderate risk of contracting grass tetany; those with more severe risk should be supplemented at a rate of two ounces per head per day.

Brucellosis

Brucellosis (Bang's disease) is a disease caused by gram-negative coccobacilli bacteria in the genus *Brucella* . Disease in cattle is typically caused by the bacterium *Brucella abortus* , although *B suis* or *B melitensis* is found in some cattle herds. Once the herd is exposed to the bacteria, it spreads rapidly, causing abortions. The disease is transmitted through the milk or through secretions from the aborted fetus and uterus. The bacteria enter the body through abraded skin, the conjunctiva, or the mucous membranes. The disease is not commonly venereally transmitted but can be transmitted by contaminated equipment. Only a small percentage of infected cattle overcome the infection. The reproductive tract may recover or the cow may shed the bacteria for life. Bacteria are present in the milk of infected cattle for a long time, typically for life.

There are two screening methods used to detect *Brucella* infection in cattle herds. The Brucella milk ring test (BRT) is the official test used to control and eradicate the disease in infected dairy herds. Every three to four months, a pooled sample of milk is tested. If the test is positive, individual cows are blood-tested; cattle that test positive on the blood test are immediately sent for slaughter. For nondairy herds, cattle are tested using market cattle testing (MCT). Serum samples are collected from cattle going to slaughter. Any reactors are traced to the herd of origin, and all animals in the herd are then tested. Any positive animals have a B brand placed on the left tail head and are immediately slaughtered. Other tests available to aid in diagnosis of brucellosis are complement fixation, serial dilution BRT, and rivanol precipitation.

Control of brucellosis is very important because this disease has public health significance. Because there is no treatment that is practical in cattle, efforts are

directed at control of infected cattle, slaughter of affected animals, and preventing the spread of the disease. Heifer calves should be vaccinated with available vaccines. The Strain-19 vaccine is a live vaccine; the RB-51 vaccine is a live vaccine but it does not stimulate antibodies detected by standard brucellosis tests. RB-51 vaccination has largely replaced the Strain-19 vaccine. Other control measures are to purchase replacement stock from a brucellosis-free area and to control stray animals. Canines can spread brucellosis when they feed on placentas and dead fetuses from infected cattle.

Vibriosis

Vibriosis is a venereal disease of cattle caused by the organism *Campylobacter fetus* sp *venerealis* . The primary clinical signs are infertility, cattle returning to estrus, cows calving later than expected, and abortions during the fifth to eighth month of pregnancy. Because these signs are nonspecific, diagnosis relies on laboratory investigation. The cows may have serum antibody titers to the bacteria. Culture of the placenta or fetal tissues reveals the organism as a curving rod with darting corkscrew-like motility. Vaginal mucus agglutination tests are used to survey herds. Infected bull penises and prepuce mucosa can be swabbed and cultured as well. Usually after five months, the infected cow recovers. Bulls may need to be treated with streptomycin injected directly into the prepuce. Vaccination is available, but does not work well in bulls.

Trichomoniasis

Trichomoniasis is caused by *Tritrichomonas foetus* , which infects the reproductive organs of cattle and causes early embryonic death. Infected bulls spread the disease to cows that remain infected for about three months. Older bulls over the age of four are generally considered to be infected for their lifetime. To treat the disease, infected cows should be sexually rested for three months or culled. There are medications that are effective against the organism, such as imidazole, dimetridazole, or metronidazole, but many are not approved for use in food animals. Infected bulls should be culled to prevent the spread of the organism. Control can also be achieved through use of vaccination and through the use of young bulls.

Anthrax

Treatment of anthrax usually is not successful in the ruminant because most animals are found dead. The bacteria, *Bacillus anthracis* , is susceptible to many antimicrobials, but therapy is usually begun too late to be effective. Meat from cattle with sudden death in an area known to have anthrax should not be consumed. Prevention and control measures should be implemented in cattle in an endemic area. Any anthrax carcasses should be cremated or buried at least

six feet deep, along with any soil or bedding contaminated with discharge. Federal officials should be notified to help contain the outbreak. The entire herd and surrounding herds should be vaccinated with the available live vaccine. The affected herd should be quarantined for two weeks after the last known death. Annual vaccination can also be given two weeks prior to herd turnout on pasture. The vaccine has a 60-day withdrawal period prior to slaughter.

Coccidiosis

Coccidiosis in cattle is primarily a disease of young cattle under a year of age. The species responsible for the clinical signs of bovine coccidiosis include *Emeria bovis* , *E zuernii* , and *E auburnensis* . This protozoan causes an acute destruction of the intestinal mucosa. The affected calves exhibit signs such as soiled rear ends and unkempt appearance and have a decreased rate of gain. Some affected calves appear to be healthy, yet shed oocysts in the manure. Others have thin, bloody diarrhea or tenesmus, go off feed, and occasionally are too weak to stand. Diagnosis is via fecal flotation, Mc Master's technique, or a direct smear of the feces to demonstrate the oocysts. Treatment in sick animals includes amprolium and sulfaquinoxaline. Prevention includes treatment of feed or water with amprolium, lasalocid, monensin, or decoquinate.

Antemortem Inspection

USDA-FSIS inspection program personnel inspect and examine all livestock presented at federally inspected slaughter plants to determine if the animal is fit for slaughter. Because not all conditions that make an animal unfit for food are noticeable postmortem, antemortem inspection must be performed. Otherwise the resulting carcass cannot get the mark of "inspected and passed." The USDA-FSIS has ruled that all nonambulatory cattle are to be condemned because this subset of animals has a significant risk of harboring the agent responsible for BSE. Nonambulatory, disabled animals include those animals that cannot walk or rise from a recumbent position. This includes those animals with metabolic conditions, broken limbs, vertebral fractures, severed ligaments and tendons, and nerve paralysis.

Postmortem Inspection Process

Under the Federal Meat Inspection Act (FMIA), food safety and inspection personnel examine and inspect the carcasses and parts of the carcass during the slaughter process. Inspection of the carcass includes the head, tongue, thymus gland, viscera, and tail. The head inspection consists of observing the head surface and eyes, observing and palpating the tongue, incising and observing the cheek muscles, and incising and observing the parotid, mandibular, medial, and lateral retropharyngeal lymph nodes. The viscera inspection consists of

observing the mesenteric lymph nodes, the abdominal viscera, the esophagus, and spleen and observing and palpating the rumino-reticular junction. The pluck (heart and lungs) is observed and palpated, the heart incised and observed, and the lung lymph nodes, including the posterior, middle, and anterior mediastinal and the right and left bronchial nodes, are incised and observed. The liver lymph nodes are incised and observed, the bile duct is opened, and the liver is palpated.

Porcine

Intravenous Injections

Pigs can be given intravenous injections using the auricular vein (marginal vein) on the ear. To give a pig an injection in the auricular vein, the pig must be held in restraint and a tourniquet should be placed around the base of the ear. The ear should be rubbed vigorously with cotton soaked in disinfection to facilitate vein dilation. Once the vein is raised, the needle can then be injected into the vein and held in place between the thumb and forefinger. The tourniquet should then be removed and the solution should then be quickly injected. A finger should be used to apply pressure to the injection site as the needle is removed.

Another route used to give an intravenous injection to a pig is via the anterior vena cava or external jugular vein. To give an injection in the anterior vena cava or external jugular vein, the pig should be held in restraint with the head raised upward and forward. The neck muscles should be stretched out and tense while in this position. The needle should be inserted at the right-sided jugular fossa, which is found lateral and cranial to the tip of the sternum. The needle should be directed toward the tip of the scapula and the operator should pull back on the syringe plunger until a flash of blood appears in the hub, which indicates the needle is in the vein. The substance can then be injected into the vein.

Performing Mucosal Layer Resection

To perform a mucosal layer resection of the rectum, a pig over 50 pounds requires a sedative and general anesthetic; in pigs less than 50 pounds, an epidural provides sufficient anesthesia. The prolapse and surrounding area should be surgically scrubbed. Two circumventing incisions should be made only in the mucosal layer: one should be around the rectal orifice and one at the proximal end of the prolapse. Connect the two incisions with a dorsal incision of the mucosal layer. Peel the mucosal layer off, ligating large vessels if necessary. Bring the two remaining mucosal layers together and suture in a simple interrupted pattern using No. 2 medium chromic gut. Place the prolapse

back into its normal position. Use a pursestring suture if deemed necessary for retention.

Rectal Prolapse

A rectal prolapse occurs when there is an evagination of the rectal mucosal tissues through the anus. It occurs in many domestic species, including cattle, sheep, pigs, and horses. It usually occurs when there is increased pressure in the abdominal cavity due to increased straining because of diarrhea, constipation, or lower intestinal inflammation. Other causes include liver disease, masses in the abdomen, irritation after rectal examination, difficult births, bladder/urethra obstruction, or chronic coughing. Other factors, such as toxins, parasites, or feeding strategies, may also play a role in the development of a rectal prolapse. The signs are fairly straightforward, with varying degrees of tissue prolapsing through the anus. Smaller and less traumatized prolapses can be reduced; larger or more traumatized prolapses require a surgical submucosal resection or reduction or resection.

Transmissible Gastroenteritis

One of the most costly diarrheal diseases of young pigs is transmissible gastroenteritis (TGE). The virus responsible for TGE is a coronavirus-transmissible gastroenteritis virus or TGEV. The virus can either occur as an epidemic in herds that are seronegative to the virus or as an endemic form in other herds. The epidemic form has a more severe clinical outcome and causes severe morbidity in piglets less than three weeks of age. Mortality rate is generally less than 20% and is attributed to the severe diarrhea leading to dehydration. Of interest is that pigs concurrently affected with porcine respiratory coronavirus (PRCV), a mild respiratory virus, will have a less severe clinical case of TGE. The endemic form of TGEV in young pigs is difficult to diagnosis and is frequently confused with other diarrhea-causing diseases.

Transmissible gastroenteritis virus (TGEV) in pigs is usually spread via the fecal-oral route. The virus needs an incubation time of 18 hours to three days before initial clinical signs occur. After the pig ingests the virus, it binds to receptors on the small intestine and is taken in by enterocytes. It then replicates in these cells and ruptures them, causing villous atrophy. While the absorptive enterocytes are destroyed, the intestinal crypts are maintained intact and in fact enlarge and continue to secrete. This sets the stage for malabsorption and maldigestion, which incite mucosal secretion and lead to severe diarrhea. TGEV is more severe in young pigs as they are not able to regenerate enterocytes or replace lost fluids as rapidly as adults.

The epidemic form of transmissible gastroenteritis (TGE) in pigs causes clinical signs, including yellowish-watery diarrhea with a foul odor, vomiting, and dehydration. In young pigs less than two weeks of age, dehydration leads to high mortality levels. Older piglets can also have less severe clinical signs, including diarrhea, anorexia, or vomiting. Lactating sows may stop producing milk. Signs are typically less severe with the endemic form than with the epidemic form. Necropsy of pigs with TGE shows a carcass suffering from dehydration; the small intestine holds a watery diarrhea and has thin, translucent walls and the mesenteric vessels are congested. Microscopic examination of the intestinal mucosa reveals a severe villous atrophy, especially in the jejunum, and a hyperplasia of the crypts.

Transmissible gastroenteritis (TGE) in pigs can be treated primarily through good nursing care. The environment should be warm (above 90 degrees) and dry. Water with electrolyte solution should be readily available to all pigs in order to rehydrate the pigs rapidly. Antibiotics may be used to combat secondary bacterial invaders; however, prevention is a better way to control the disease. In a seronegative herd, additions should be only from seronegative herds; even then, these additions should be quarantined for a month and retested before placing them into the herd. Strict biosecurity measures should be implemented for all personnel, and all potential vectors, such as mice, birds, cats, dogs, and rats, should be kept from the hog building. Control in infected herds relies upon sows acquiring IgA and passing this immunity on to their nursing piglets.

Swine Dysentery

Swine dysentery is caused by *Brachyspira hyodysenteriae* and is highly contagious. The spirochete multiplies in the large intestine and causes a bloody, mucous-laden diarrhea through inflammation, edema, and damage to the intestinal mucosa. Pigs of all ages are susceptible to this common disease. Clinical signs begin as fever, anorexia, and soft stools that turn into a mucohemorrhagic diarrhea. The diarrhea and intestinal damage cause the affected pigs to become dehydrated. Treatment is with antibiotics, being certain to test because resistance to certain antibiotics has occurred. Control is important, as the disease can become endemic in herds. Depopulation, followed by disinfection and repopulation with disease-free pigs, may help prevent herd disease; rodent control may also be useful because rodents may harbor the spirochete.

Ovine/Caprine/Cervidae

Q Fever

Q fever is a disease caused by the rickettsia *Coxiella burnetii.* This agent causes abortion in sheep and goats and is infectious to other animals, including humans. Because Q fever can cause abortions in pregnant women, they should not handle Q-fever infected animals or their tissues. Other than causing abortions, the disease goes largely unnoticed in a flock of sheep or goats. Abortions occur late-term or the ewe may deliver a weak lamb. The placenta of a Q fever-aborted lamb appears thickened with white, chalky plaques and a reddish-brown exudate; the tissues have a high number of neutrophils and rickettsia. The ewe will also have a high titer to the organism. Tetracycline kills the organism, which is carried by ticks or through contaminated feed and water.

Progressive Pleuropneumonia

Ovine progressive pleuropneumonia (OPP) is a chronic disease of sheep. Only 20% of sheep infected with the virus develop clinical signs and usually these sheep are over two years of age. The signs include chronic pneumonia, rapid breathing, expiratory dyspnea, abdominal breathing, lack of a fever, and loss of body conditioning despite good appetite. Pregnant ewes may have lambs that are weak or small. AGID is a common screening test and ELISA tests are also available. There is no treatment for OPP or any vaccination. Control is based upon testing flocks, culling of infected animals, removal of lambs from infected ewes before colostrum is consumed, or feeding lambs pasteurized colostrum and milk from infected ewes.

Intravenous Injections

Sheep and goats are most commonly given intravenous injections into the jugular vein. The animal is restrained while standing or while lying on its side. The head should be moved to the side to expose the neck and to tense the neck muscles. If the hair coat is thick, a shear should be used to clip the wool or hair away from the jugular groove about midway down the neck. Using the fingertips of the non-dominant hand, pressure should be applied to the jugular groove proximal to the site of injection to raise the vein. The needle is then inserted and threaded into the vein, making sure blood is flowing freely. Fingertip pressure is removed from the jugular groove and the substance is injected.

Ewe and Doe Pregnancy Toxemia

Pregnancy toxemia is a condition affecting ewes and does during the last two to four weeks of pregnancy. It is caused by the negative energy balance that occurs when the growing energy demand from the fetus outstrips the energy intake from the dam. Dams carrying more than one fetus, overweight ewes, or those fed poor quality feed are commonly affected. An affected animal may be weak,

go off feed, become depressed, hide away from the herd, appear blind, and will eventually become recumbent. Some ewes and does have neurological signs such as grinding of the teeth, circling, ataxia, tremors, or star-gazing. Ketonuria and ketonemia are increased; the animal may have acidosis as well. Treatment is to remove fetuses either by inducting parturition or by cesarean section. The ketosis must also be treated using IV therapy combined with B vitamin administration along with propylene glycol given orally.

Scrapies

Scrapies is a progressive and fatal disease of sheep that causes encephalitis. The responsible agent, a prion, is resistant to most chemical or environmental treatment. Clinical signs of the disease in sheep occur after a long incubation period (one to seven years). In the early stages, the sheep withdraws itself from the rest of the flock, may act aggressive or nervous, and experiences itching. The itching can be intense, causing the sheep to bite and lick the skin until the wool falls off and the skin is damaged. If the itchy areas are scratched, the sheep will nibble, lick, or grind its teeth. As the disease progresses, the animal develops anorexia, ataxia, tremors, blindness, abdominal distention, and loses weight. The animal will die anywhere from six weeks to one year after developing clinical signs either from convulsions or starvation.

Control of scrapies is important as there is no treatment for this disease. Sheep that display clinical signs should immediately be destroyed and the herd mates should be euthanized or the premises quarantined for two years. This disease is reportable and the affected animals and progeny should be destroyed. Pastures that have had scrapies-positive animals should not be used for livestock. Buildings and barns that have held sheep with scrapies should be thoroughly cleaned of organic material, disinfected, and kept free of livestock. Sheep producers should keep accurate production and medical records so that traceback of infected animals can be performed. Euthanized animals should be properly disposed of and regulatory personnel should be notified to ensure proper disposal.

Caseous Lymphadenitis

Treatment for caseous lymphadenitis is not generally successful. *Corynebacterium pseudotuberculosis* is susceptible to the penicillin group; however, because abscesses generally reform after lancing and draining, the drug has limited ability to rid the animal of the organism. The herd owner should practice culling of infected animals, use of vaccination, and environmental clean-up. Animals with abscesses should be isolated from those without and culled if possible. If an infected doe has a kid, it should be

immediately removed to prevent spread to the kid. Pens should be maintained so that there are no objects that can cause punctures or wounds. Any equipment should be disinfected before and after use between animals. External parasites should be controlled so that animals with itching do not rub themselves on pens or walls.

Reproductive Cycle of Sheep

Sheep have 54 chromosomes and breed throughout the year, although there is some dependence on photoperiods. Most sheep breed in decreasing or short day lengths; domestic sheep have breeding seasons from five to seven months. The estrus cycle averages about 16 days. Estrus is influenced by the presence of the ram and lasts for about 30 hours. After the egg is fertilized, it begins dividing and after about 17 days, attaches to the endometrium. The placenta is diffuse for about 30 days after attachment; however, it becomes a polycotyledonary placenta with cotyledons on the placenta and caruncles on the uterus, forming the placetomes. The average length of gestation is 148 days.

Equine

Estrus Cycle

The estrus cycle in mares can be variable. A small percentage of mares are polyestrous and will cycle on a regular basis throughout the year. Other mares are seasonally polyestrous. These mares have a cyclic period and then an anestrous period of no cyclical activity, usually in the winter. The mare will ovulate, then have a regular cyclical period, and will stand to be bred by the stallion for time periods of two weeks to two months. The mare in anestrous will remain so for a variable period of time from a little over a month to up to eight months. Another group of mares considered to be seasonally polyestrous also have irregular reproductive patterns. These mares have irregular responses to being teased and may go into heat without ovulation or vice versa.

There are a number of behaviors displayed by a mare in estrus. This behavior is best demonstrated when the mare is exposed to a stallion. Teasing is the exposure of the mare to a stallion and should be done every day during the breeding season. Sometimes mares will need to be teased by different stallions to elicit the estrus behavior. The safest way to tease is through use of a teasing rail. Mares in estrus that are exposed to a stallion will display the following behaviors: standing, squatting, and everting the vulval labia (wink), raising the tail, and urinating. Mares that are not in estrus become agitated when a stallion is presented to them; they may squeal, kick, or lay their ears back.

Foal Rejection

Arabian mares and first-time mothers are more likely to reject their foal than other breeds of horses. Mares that are restrained so that they cannot harm the foal are likely to let the foal nurse. Foal rejection is thought to have a genetic basis, or may be due to the mare's temperament, environmental disturbances (human interference or the presence of other animals), or poor health of the mare (dystocia or exhaustion). A rejection can consist of walking away or ignoring the foal or it can be as severe as kicking at the foal as it tries to nurse. The udder should be inspected for signs of mastitis or trauma. If the udder is normal, the mare should be restrained while the foal nurses, using partitions or by tying the mare's head to prevent nipping or holding one leg up to prevent kicking. Because some mares will allow hand milking, this technique may be used to get the mare used to having the teats manipulated.

Red Maple Poisoning

Red maple poisoning occurs in horses that are pastured in areas where they have access to red maple trees (*Acer Rubrum)* . The toxin that causes the acute hemolytic anemia is not known at this point, but wilted or dried leaves are particularly toxic. The signs of red maple poisoning are attributed to methemoglobinemia and Heinz body formation and include cyanosis, anorexia, depression, icterus, hemoglobinuria, anemia, and dehydration. The blood also takes on a brown discoloration. The signs can be similar to other diseases such as babesia infection or equine infectious anemia or similar to poisoning by nitrates, onions, or phenothiazine. Treatment includes IV fluids (added bicarbonate may be necessary) and blood transfusions. Prevention is based upon restricting the horse's access to the leaves and trees.

Septic Arthritis

Septic arthritis arises in the equine from three primary routes. In the foal, the navel can become infected and spread bacteria from the infected navel to joints. Bloodborne joint infections can also be caused by intestinal or lung infections. Bacteria can also be introduced through skin or foot trauma directly into the joint or the surrounding tissues. The third primary cause is iatrogenic following a joint injection or through a contaminated surgery site. The treatment for septic arthritis should be aggressive through culture and sensitivity testing of joint fluid to direct therapy with the appropriate systemic broad-spectrum antibiotic. Generally, joint lavage, debridement, and drainage are indicated, along with intra-articular antibiotics to achieve healing. Usually, an appropriate nonsteroidal anti-inflammatory drug (NSAID) is given to relieve inflammation and to achieve pain control.

Gastric Ulcers

Gastric ulcers are relatively common in the horse. Half of all foals and one third of adult horses have mild gastric ulcers. The majority of race horses are also afflicted with gastric ulcers. Most cases are mild and not suspected and heal without treatment. The nonglandular squamous mucosa is the most common site for gastric ulcer formation, followed by the antrum and pylorus of the glandular mucosa, then the glandular mucosa corpus. Many cases of gastric ulcerations have no known causes. Some theories proposed for the development of ulcers include delayed feeding (equines continuously excrete hydrochloric acid in their stomachs), heavy exercise regimes, heavy use of NSAIDs, infection of the stomach with *Helicobacter* , or delayed gastric emptying.

Infectious Anemia Virus

Coggins testing, an agar-gel immunodiffusion test, is used to identify equine infectious anemia virus (EIAV) carriers. It is the "gold-standard" serologic test and is used extensively to control the spread of the disease. Interstate transport regulations, shows, and fairs regularly require a negative test in order to engage a horse in these activities. If a horse is found to be positive, there are general control measures to follow to prevent the carrier from transmitting the virus. After testing positive, the horse must be segregated from negative-testing horses and kept stabled in an insect-proof or -resistant environment, and no equipment is to be shared between the carrier horse and other horses. All horses without a negative Coggins test should be considered possible carriers of EIAV.

Sarcocystis Neurona

One possible explanation on how *Sarcocystis neurona* causes damage to the horse's CNS is as follows. After the horse ingests the sporocysts, these sporocysts release sporozoites in the intestine. The sporozoites then burrow through the intestine and enter the endothelial cells of arteries. While in the endothelial cells, schizonts develop and rupture merozoites, which are released into the bloodstream. This is a critical point, as the horse's body can clear the *S. neurona* infection and only become seropositive for the organism. If the body does not clear the infection, the merozoites potentially enter the CNS through infected leukocytes or through endothelial cytoplasm. Once in the CNS, the merozoites form schizonts, which go on to form more merozoites. At whatever site in the CNS this occurs, the lesions produce clinical signs.

Tetanus

The treatment for a horse suffering from tetanus centers on combating the toxin, controlling the pathogen, relaxing the muscle spasms, and providing good

nursing care. Despite treatment, there is a high mortality rate. Wounds should be cleaned and necrotic tissue removed, and if needed, drained. Penicillin should be given to combat the active pathogen, *Clostridium tetani* . To combat the toxin and its systemic effects, tranquilizers, sedatives, and/or curariform medications should be used, along with 300,000 IU of tetanus antitoxin twice daily. Another option is to inject 50,000 IU of tetanus antitoxin into the subarachnoid space via the cistern magna. Nursing care should consist of keeping the horse in a darkened stall and keeping the area quiet, using cotton in the ears if necessary. Water and feed should be available and placed in such a position that the horse does not need to lower its head.

Navicular Disease

Navicular disease is a common cause of lameness in horses. This chronic disease involves the navicular bone and navicular bursa degeneration. Since it is chronic and an ongoing process, the disease cannot be cured. Rather, it is managed with rest, nonsteroidal anti-inflammatory drugs (NSAIDs), ongoing foot care, injection of corticosteroids, and in extreme cases, palmar digital neurectomy or severing of the collateral sesamoidean ligament. Proper hoof trimming and shoeing is essential. The hoof should be trimmed to make sure the foot bones are properly aligned and the toes should be rounded. Corrective shoeing to give more support to the heel should be used. An egg-bar shoe made out of aluminum will be lighter than a regular horseshoe. NSAIDs, isoxsuprine hydrochloride paste, and hoof moisturizers will help alleviate pain, increase blood flow to the effective area, and further protect the hoof.

Idiopathic Hemiplegia

Idiopathic Hemiplegia, or roaring, is caused when damage occurs to either one of the two recurrent laryngeal nerves. This damage causes a unilateral paralysis of the larynx, particularly the arytenoids cartilage and vocal fold. Why this damage occurs is usually unknown, although cases of strangles have caused this paralysis. There may be a heredity component to the disease, or it may be due to anatomy. The left recurrent laryngeal nerve is typically involved, which is longer than the right. Long-necked, tall male horses seem predisposed. Affected horses have difficulty with exercise, and while exercising, a distinct “roaring” noise is heard during inspiration. Treatment is a combined laryngoplasty/ventriculectomy to remove laryngeal tissue and to tack down the remaining tissue. While this will usually improve airflow, complications may arise such as a chronic cough or infection at surgery site.

Sarcoids

Sarcoids are common soft-tissue tumors of horses. They can vary in shape, size, and appearance and some cases are very difficult to treat. Many different treatment options have been tried. Surgery to remove the tissue is a common treatment, but does have a high rate of recurrence; about 50%. Autogenous vaccinations, use of immune stimulants, laser removal, application of medicated creams, and cryotherapy have been used with varying amount of success. For refractory cases, chemotherapy drug injection into the sarcoid has been successful. This treatment is typically performed over a period of two months or longer. Radiation therapy is usually successful. Radioactive seeds can be planted into the sarcoid, although this does require radiation safety precautions due to continuously emitted radiation, or external beam radiotherapy can be used to diminish exposure to radiation.

Paraphimosis

Paraphimosis is the inability to withdraw the penis back into the prepuce. This occurs due to strictures of the prepuce with which the stallion can be born or acquire due to trauma or disease. Other causes of paraphimosis in the stallion include balanoposthitis, paralysis of the penis, use of phenothiazine tranquilizers, or injury to the penis, which makes retraction into the prepuce impossible. If the condition has recently occurred, the penis can be returned to the prepuce by generously lubricating the penis and prepuce and manipulating the penis back into the prepuce. In cases where the penis has been allowed to remain outside the prepuce to the point that it has become swollen, surgery to enlarge the preputial orifice may be necessary to replace the penis. Other treatment options include ice packs, cold water baths, anti-inflammatory agents, diuretics, and protecting the penis through ointments and support.

Parascaris Equorum Infection

Parascaris equorum infection is more common in foals than in adult animals. Foals become infected through environmental contamination of pastures, corrals, and stalls by the eggs, which can remain viable in the soil for years. The adult *Parascaris equorum* worm is a white worm with three lips that can reach lengths of up to 30 cm. It has a prepatent period of 10-12 weeks. Intestinal infection produces signs of lethargy and an unkempt appearance due to nutritional competition. In heavy infestations, *Parascaris equorum* can produce signs of colic, intestinal obstruction, or perforation. Occasionally, migrating larva can pass through the respiratory tract and produce signs of verminous pneumonia. Diagnosis is made through clinical signs and fecal examination to identify eggs. Any broad-spectrum anthelmintic is effective against the adult and immature worms. Respiratory-related infestation can be treated with ivermectin or fenbendazole, along with a broad-spectrum antibiotic.

Strangles

Streptococcus equi equi (gram-positive organism) is the cause of strangles in donkeys, horses, and mules. These are the only species that contract disease from these bacteria. The highly contagious bacteria are spread via fomites and direct contact of a noninfected horse with the exudates from an infected horse. The bacteria can live for up to two months in the environment, provided acceptable conditions exist. Sunlight, dry weather, and heat can kill the bacteria. Natural disease does give a lengthy period of protection against strangles. Control of the disease is accomplished through isolation of sick animals for at least one month and use of strict biosecurity measures, such as disinfection, fly control, and clothing changes. All animals exposed to sick animals should be carefully scrutinized for signs of the disease and isolated as needed. Before releasing isolated animals, three negative cultures of the nasopharynx should be obtained four to seven days apart.

Preventing Equine Influenza

The cornerstone of preventing a horse from suffering the effects of equine influenza is vaccination. Horse owners should be encouraged to adopt a vaccination schedule to prevent the virus from rendering their animal unfit for competition or work. The virus can sideline a horse for up to three months. After the initial vaccination, the vaccination should have a booster in three to four weeks. In young horses, a vaccination schedule every four to six months should be followed in order to maintain adequate antibody levels. Older horses that have been regularly vaccinated should have a booster shot once a year in order to maintain adequate antibody levels. Colostrum in mares adequately vaccinated can provide protection to the foal. If mares are vaccinated twice a year, the foal should not receive its first vaccine until after it is six months old to prevent maternal antibody interference.

Routine Dental Maintenance

After a routine examination of the oral cavity of the horse, various issues with the dentition can arise. Many horses need to have their teeth floated in order to correct wear patterns that may interfere with eating or use of the bit. Primarily, this is done to remove sharp tips or hooks of the molars. It is very important to have the proper equipment and adequate restraint in order to inspect and to use equipment in the mouth. Mild chemical restraint is generally used, along with either a mouth speculum, dental wedge, and/or a dental halter. Special attention should be paid to any wolf teeth present (removal might be needed) and any retained dental caps. Usually, canine teeth cause no problems, but they

may need to be ground down in males. The incisors should also be inspected for problems.

Preventing Encephalitis

These viruses are spread through a combination of insect vectors and intermediate hosts, such as birds, small mammals, and reptiles. Control of these will help prevent the spread of the viruses to susceptible horses. Use of insect repellents on the horse, drainage of standing water around the premises, prompt manure removal and adequate disposal, chemical control around property during time of heavy infestations, and use of screens on buildings are all ways to control the insects. Proper feed storage, cutting of grasses, and building maintenance will prevent attraction and access of mammals and reptiles to horse-congregating areas. Vaccination is mandatory in addition to control of vectors and hosts. Foals should be vaccinated at three, four, and six months of age and then twice a year. Pregnant mares should be vaccinated three or four weeks before foaling.

Parasite Control Program

Parasite control programs need to be tailored to the prevalence of the parasite on the pasture, pasture stocking density, and age of the horse. Older horses can become resistant to certain parasites, such as *Strongyloides westeri* and *Parascaris equorum* . The main digestive tract parasites of the horse are the nematodes. In general, practitioners work with three main classes of anthelmintics: benzimidazoles, avermectins/milbemycins, and pyrimidines. Benzimidazoles kill most nematodes. Avermectins control nematodes as well as external parasites such as bots, ticks, lice, and mites. Mibemycins treat cyanthostomes. The pyrimidines are effective against tapeworms in addition to nematodes. An effective anthelmintic schedule should routinely shift which type of anthelmintic is used, taking into account the most likely type of parasite the horse could be harboring.

Recurrent Uveitis

Equine recurrent uveitis is also known as moon blindness. While the exact cause is not known, authorities suggest it may be a local immune response to pathogens such as bacteria, viruses, or parasites. It manifests as recurring episodes of acute uveitis followed by periods of apparent normalcy of the eyes. After a number of episodes, other eye structures become damaged, which can lead to blindness. When a horse has an episode, the eye becomes painful and blepharospasm, redness of the conjunctiva, and tearing occur. The eye may also appear to be cloudy. The treatment for equine recurrent uveitis revolves around decreasing inflammation. Corticosteroids, nonsteroidal anti-inflammatory

drugs (NSAIDs), atropine, and ophthalmic ointment antibiotics have all been used to treat this disease condition.

Treating Ulcers

The choice of therapeutic agents used to treat both gastric and duodenal ulcers in the horse and foal is based upon which agents treat the underlying cause and maintain a gastric environment that helps heal ulcers. Generally, this is achieved through suppressing stomach acid and protecting the mucosa. There are four general classes of medications that achieve these goals: Mucosal protectants, primarily sucralfate, bind to the ulcer and stimulate mucous secretion; antacids, such as Amphojel, Maalox, Mylanta, and Riopan, briefly decrease gastric acid. Proton pump inhibitors such as omeprazole are potent drugs that totally block gastric acid secretion by inhibiting the hydrogen ion pump. Histamine receptor antagonists, such as cimetidine, famotidine, and ranitidine, are commonly used to inhibit gastric acid secretion in the horse.

NSAIDs Precautions

Nonsteroidal anti-inflammatory drugs (NSAIDS), particularly flunixin, meglumine, and phenylbutazone, are commonly used in the horse to treat acute and chronic inflammatory processes. At recommended dosages, they are generally well tolerated. Increased doses can lead to gastrointestinal and renal complications. All these agents generally work by inhibiting prostaglandin synthesis by inhibiting cyclooxygenase, an enzyme which converts arachidonic acid into endoperoxides, which in turn are converted to prostaglandins. In the digestive system, prostaglandins protect the stomach mucosa. NSAIDs, especially in high doses, disrupt this protective effect, leading to signs of NSAID toxicity in the digestive tract, including anorexia, abdominal pain, and diarrhea, and can damage the renal medulla and pelvis, leading to blood and increased protein in the urine, azotemia, and disturbances in fluid concentration.

Function of Large Intestine

The horse's large intestine functions to store and absorb large amounts of fluid. As much as half of the horse's food is digested via microbes in the large intestine (hind gut fermentation). The small intestine empties into the cecum at the ileocecal junction. The cecum has an average length of one meter and can hold about 33 L of ingesta. It has four longitudinal bands: the cecocolic fold, the ileocecal fold, and a ventral and medial band. The ascending colon is approximately three to four meters long and can hold an astounding 130 L of ingesta and fluid. It is only attached to the body wall at the right ventral and the right dorsal colon. The ascending colon is considered to be the large colon and is composed, in order from the cecum, of the right ventral colon, the sterna

flexure, the left ventral colon, the pelvic flexure, the left dorsal colon, the diaphragmatic flexure, the right dorsal colon, and the transverse colon. The transverse colon then merges as the small colon, which empties into the rectum.

Meconium Retention

Meconium retention can create an obstruction in the neonatal foal. The impaction is formed from meconium composed of amniotic fluid, bile, epithelial cells, and intestinal secretions. The signs exhibited by the foal depend upon the degree of obstruction and can include poor appetite, abdominal discomfort, restlessness, straining, and tail elevation. Signs of colic due to gas accumulation proximal to the impaction can occur. Diagnosis is based upon these signs, as well as failure to pass stool, physical examination, and occasionally, radiographs. Initial treatment is based upon repeated enemas consisting of acetylcysteine to soften the meconium, warm water, mineral oil, or dioctyl sodium sulfosuccinate (DSS). If this fails to relieve the impaction, surgical intervention may be needed.

Liver Failure

Treatment for horses with liver failure is through good supportive care and medical treatment. Priorities are to first treat any encephalopathy and acute liver failure. Horses with seizures or that are extremely agitated should be sedated, keeping in mind that because the liver metabolizes most sedatives, dosing must be done at minimal levels. Diazepam should be avoided, as it may worsen the signs. Antibiotics are needed if the liver failure is caused by a bacterial infection. Acidosis may be a problem and must be slowly corrected with bicarbonate. Low blood glucose levels should be treated with IV glucose administration. High blood ammonia levels can be decreased through use of a mild laxative and oral neomycin or lactulose. Metronidazole also decreases ammonia-producing bacteria. When the horse can eat, it should be fed small meals with balanced protein and energy levels.

Review Video: Liver Failure
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Urinary Tract Infections

Urinary tract infections in horses are commonly caused by an underlying neurological condition, urolithiasis, from attempts at catheterization, and damage to the urethra. Some bacteria that have been found to cause urinary tract infections in the horse include: *Escherichia coli* , *Proteus mirabilis* , *Staphylococcus* spp, *Klebsiella* spp, *Corynebacterium* spp, *Enterobacter* spp, and

Pseudomonas aeruginosa . Treatment of a horse with a urinary tract infection should be based upon culture and antibiotic testing to determine the causative agent and most effective antibiotic. Antibiotics commonly used in urinary tract infections include amoxicillin, ampicillin, penicillin, trimethoprim/sulfadiazine, nitrofurantoin, and amoxicillin. The aminoglycosides, amikacin, and gentamicin, can also be effective but they can cause kidney damage.

Lumbosacral Spinal Tap

Cerebrospinal fluid (CSF) can be collected in the horse via a lumbosacral spinal tap or by tapping the cistern magna. Cerebrospinal fluid is produced in the choroid plexuses of the lateral, third, and fourth ventricles, the ependymal lining of the ventricular system, the pia arachnoid, and the meningeal blood vessels. To collect CSF from the lumbosacral cistern, the horse should be slightly sedated and the skin over the area of L6 to S1 should be clipped and surgically scrubbed. The skin is anesthetized with lidocaine over the depression between L6 and S1. A one-centimeter incision is then made through the skin at this site. Using an 8-inch, 18-gauge spinal needle, the needle should be inserted into the incision until the tip punctures the lumbosacral cistern. The jugular vein should be compressed to increase CSF pressure, and fluid will begin to drip from the needle hub.

Herpesvirus Encephalomyelitis

Diagnosis of a horse suspected to have herpes viral encephalomyelitis is made by demonstrating a fourfold or more rise in titer. The cerebrospinal fluid of horses displaying signs of herpes viral encephalomyelitis can also be examined for antibodies against equine herpesvirus type 1. The virus causes a vasculitis in the central nervous system tissue, followed by thrombosis; immune complexes are formed as well. Hemorrhage and edema occur in the brain, leading to clinical signs. Treatment is largely supportive for horses suffering from herpes viral encephalomyelitis. Corticosteroids such as dexamethasone should be given twice a day for the first two days, along with concurrent use of broad-spectrum antibiotics to guard against secondary bacterial infections. Animals with bladder dysfunction should be catheterized. Fluid and food intake should be carefully monitored and therapy should be initiated in those animals unwilling to eat or drink.

Epiploic Foramen Entrapment

Epiploic foramen entrapment is more likely to occur in horses over six years of age. The epiploic foramen (foramen of Winslow) separates the omental bursa and the abdominal cavity in the horse. The ventral boundaries are the gastropancreatic fold, the portal vein, and the pancreatic right lobe. The dorsal

boundaries are formed by the caudate lobe of the liver and the vena cava. The caudal boundary is the intersection of the mesoduodenum and the pancreas, while the cranial boundary is the hepatoduodenal ligament. A left to right displacement of the omentum may result from it being pushed by the small intestine through the epiploic foramen, or the intestine itself may come through. More commonly, the intestine passes from right to left into the omental bursa.

Mature horses over six years of age are more likely to suffer from epiploic foramen entrapment. These horses are more likely to have a smaller liver, which increases the size of the epiploic foramen, leading to entrapment of the small intestine and/or omentum in this opening. The opening is a natural opening formed by the caudate lobe of the liver, the inferior vena cava, and the portal vein. This can be a difficult disorder to diagnose, as affected horses may have vague signs. The horse may act depressed or have abdominal pain, gastric reflux and/or abdominal distention. Frequently, any peritoneal fluid obtained prior to surgery appears normal. Many times the diagnosis will be made at surgery. The entrapped intestine will have to be exteriorized, examined for defects, and if needed, an anastomosis must be performed. Prognosis depends on the amount of devitalized tissue and how soon the condition was treated.

Rectal Lacerations

A rectal laceration or tear through the rectal or anal mucosa occurs when foreign bodies or damage from rectal palpation occur. The horse suffering from a rectal laceration may have difficulty defecating or have constipation. The animals may also have tenesmus, hemorrhage, and swelling in the perianal area. Treatment for a rectal laceration should occur as soon as the rectal tear is diagnosed. The perianal area should be cleansed and the horse should be given broad-spectrum antibiotics and IV fluids with or without flunixin; meglumine should be started if the animal is severely injured. Mineral oil can be given via a nasogastric tube to soften the feces and the horse should be fed a laxative-type diet, such as pasture grass or alfalfa hay. Grade 1 tears can be treated with medical treatment only; grade 2 and grade 3 tears require surgery. Grade 4 tears carry a grave prognosis despite treatment.

A rectal laceration or tear in a horse is classified according to the tissue layers that are torn. A grade 1 tear involves only the mucosa layer of the rectum, while a grade 2 tear involves tearing of the muscular layer only. A grade 3 rectal tear involves the mucosa, submucosa, and the muscular layers. This grade is further divided into grade 3A, which means that the serosal layer is the only layer intact. A grade 3B laceration is a dorsal laceration into the fat of the mesocolon. A grade 4 laceration is a tear that disrupts all the rectal layers and permits entry

of contamination into the abdominal cavity. Grade 1 tears can be treated conservatively with fecal softeners, broad-spectrum antibiotics, flunixin, meglumine, and rest. Grade 2 and grade 3 rectal lacerations will need medical treatment combined with surgery. Grade 4 lacerations have a dire prognosis.

Guttural Pouch Tympany

Young foals can suffer from tympany of the guttural pouches. When this occurs, either the guttural pouch or both pouches becomes distended with air. The foal afflicted with guttural pouch tympany has a soft fluctuant swelling in the retropharyngeal space. This swelling does not cause the foal any pain, but causes varying degrees of respiratory distress, difficulty with swallowing, and extension of the head and neck. To relieve the air distention, the skin can be punctured with a needle at the Viborg's triangle or a catheter can be passed through the pharyngeal opening of the guttural pouch; however, this is only temporary. Surgery to remove redundant plica salpingopharyngea, or if only one pouch is affected, fenestration of the septum between the two pouches, permanently relieves the problem.

Guttural Pouch Empyema

In guttural pouch empyema, pus accumulates in the guttural pouches secondary to other guttural pouch infections or due to upper respiratory tract infections. It is often unilateral and frequently follows an infection with *Streptococcus equi* . The horse has continued drainage of white opaque discharge from the nasal passages after it recovers from an infection elsewhere. Abscesses can also rupture into the guttural pouches and lead to the nasal discharge. In addition, the horse may have swelling of the parotid lymph node, respiratory distress, and difficulty with swallowing. Radiographs, needle biopsy of the guttural pouch, and endoscopic examination are all useful diagnostic techniques used to diagnose guttural pouch empyema. Medical treatment consists of antibiotics along with daily lavage of the guttural pouches. Surgery to drain the pouches may be necessary.

Osteochondrosis

Treatment of a horse with osteochondrosis (OCD) can either be medical, surgical, or a combination of the two. Medical treatment includes diet and rest. The disease is thought to be a combination of heredity, hormonal, and dietary factors. Animals fed for a high weight gain are more prone to OCD. High zinc levels and dietary copper deficiency may play a role in the development of OCD. Horses with OCD have abnormal cartilage growth. The cartilage cells fail to differentiate normally and cartilage can become abnormally thickened. If medical therapy is not successful, surgery can be attempted. Surgery is

indicated to remove loose cartilage flaps, fragments of cartilage or bone in the joints, or to remove bone cysts.

Angular Limb Deformity

A foal suspected of having an angular limb deformity should have strict stall rest until radiographs can be taken to ascertain the degree of deformity. If the angle of deformity is less than 10 degrees and if the bones are undergoing normal ossification, the foal can be treated conservatively with stall rest and controlled exercise until the structures that support the growth cartilage are more fully developed. Corrective hoof trimming can also be implemented as the foal grows. If the foal has a more severe angle of deformity, casting, splinting, or bracing may be of benefit. If the foal does not respond to any of these measures, surgery may be warranted. Modulating the growth of the physis can be done by use of bone plates and screws or by a wedge osteotomy. Periosteal transection (stripping) can be done to accelerate bone growth on the concave side of the bone.

Severed Tendons

Due to their superficial location and scant muscle, the distal limb is prone to laceration or severing of the tendons. This is usually the result of trauma, quite typically due to fence wire. If the flexor tendons are severed, the horse will not be able to bear weight on the affected appendage. If the extensor tendons are severed, the horse will be able to bear weight on the limb but it will drag its toes or knuckle over. Treatment for severed tendons is always surgical. Severed extensor tendons must be surgically debrided and the ends of the tendons sutured together. Casting for four to six weeks after surgery and stall rest will typically give good results. Severed flexor tendons are treated in a similar manner as severed extensor tendons, but the prognosis for return to function is poor.

Positioning Fetus During Parturition

The horse fetus is quite mobile during the third and fourth month of gestation. As the end of gestation approaches, the fetus begins to orientate itself in the normal foaling position, with its head and front limbs pointed toward the cervix. The hindlimbs become enclosed in the other horn and can be oriented such that the hindlimbs rest on the dorsal surface of the uterine body, and possibly, even over the fetal head. During labor, the head and forelimbs become fully extended. When the fetlocks are at the external cervical opening, the placenta ruptures. As the head appears at the vulva, the withers rotate in a dorsosacral position. As the hips exit the vulva, the fetal pelvis rotates through a dorsoilial position into a dorsosacral position.

Third-Degree Perineal Laceration

Prior to repair of a third-degree perineal laceration, the mare should be placed on a diet that will soften but not liquefy the stool, such as pasture grass or alfalfa. The mare should also be started on antibiotics and should have been given a tetanus injection at the time of injury. Third-degree perineal lacerations involve tearing through the rectum, vagina, perineal body, and the rectovaginal septum. The repair is done in a two-stage operation to avoid straining and failure of the reconstruction. In the first stage, the tear between the vagina and the rectum is repaired. The second stage occurs when the perineal body is reconstructed. Two to three weeks of healing time should be given between each repair.

Pneumovagina

Some mares develop pneumovagina, which is the noisy aspiration of air into the vagina. This is caused by trauma due to foaling injury or breeding injury or conformational conditions. Mares that have a large vulva opening, older mares, mares that are underweight, or those that have a sunken anus are also more likely to have pneumovagina. The condition can cause problems with fertility due to aspiration of fecal material. Infection with fecal bacteria leads to vaginitis and the development of cervicitis and endometritis. Pneumovagina can also lead to urine pooling in the vagina. If the mare does become pregnant, the placenta or uterus can become infected by fecal bacteria, leading to loss of conception or abortion of the fetus.

Caslick's Operation

A Caslick's operation is performed on mares and involves partially suturing the lips of the vulva together (a vulvoplasty) in those mares that have an abnormal vulva conformation. A normal mare should have a vulva with full and firm lips. The lips should meet evenly on the midline, and 80% or more of the opening of the vulva should be below the brim of the pelvis. If more than four centimeters of length of the vulva seal is dorsal to the pelvic floor, the seal is abnormal and air and/or fecal matter can enter the vagina. The Caslick's operation involves restraining the mare in stocks and giving her a sedative. Local anesthetic is infiltrated at the mucocutaneous junction of the vulva. A narrow strip of mucosa is then removed from the mucocutaneous junction, and the exposed submucosa is sutured together.

Castration

Castration in a male horse is usually done after the horse is a year old. Both testicles should be descended to perform a simple castration. The horse can be

castrated either standing or in recumbency. Standing surgeries are done using a sedative/tranquilizer along with local anesthesia in the testes and spermatic cord. When the horse is recumbent, general anesthetic is used, and one of the legs is tied up to give access to the surgical site. Each testis is incised separately; the skin, tunica dartos, and scrotal fascia are incised, leaving the tunica vaginalis parietalis (common tunic) intact. The subcutaneous tissue is stripped from the common tunic before it is incised. The emasculator is applied to the spermatic cord and associated structures, including the external cremaster muscle, common tunic, and ductus deferens. The incision is left open to heal and a tetanus vaccination is given.

After a male horse has been castrated, there are complications that can arise. The newly castrated horse should be kept away from mares for a week to prevent pregnancy. Persistent stallion behavior can persist despite complete removal of the testicles and associated tissues. An inguinal hernia can develop leading to intestinal contents spilling out the surgical site. Excessive hemorrhage can occur from the testicular artery of the spermatic cord or branches of the external pudendal vein. A scirrhotic cord can form after infection. The surgical site can swell or a hydrocele can form due to fluid collection because of poor drainage or entrapment of fluid in the common tunic. The wound itself can become infected and the gelding can develop septicemia. Proper attention to technique, making sure the incision has good drainage, and good nursing care postoperatively minimize the risk of these complications.

Emergency Tracheostomy

A horse may need an emergency tracheostomy due to conditions that obstruct the airway such as abscesses, neoplasia, pharyngeal or nasal swelling or edema, or guttural pouch distention. Nonemergency conditions may also require the veterinarian to perform a tracheostomy. After the standing horse has been sedated, the hair over the surgery site (usually middle third of the neck) is surgically scrubbed, then local anesthetic is infused subcutaneously along the surgery site. The skin, subcutaneous tissues, and cutaneous colli muscle are incised. This exposes the paired sternothyrohyoides muscles, which are bluntly divided at midline. The tracheal rings are thus exposed and a scalpel is used to make an incision in a horizontal direction. The opening should be just wide enough to pass the tracheostomy tube.

Perineal Urethrotomy

A male horse may need a perineal urethrotomy if the urine is blocked from leaving the body due to an obstruction such as a hematoma, uroliths, granulomas, or a torn urethra. The surgery is performed while the horse is

standing. Sedation should be given along with an epidural anesthetic. During the surgery, the retractor penis muscle, the bulbospongiosus muscle, corpus spongiosum, penis, and urethra are all incised. Complications from this procedure include excessive bleeding, stricture formation, urine scald, incontinence, orchitis, or peritonitis. If the procedure was performed because of an obstruction of a urolith, the condition may recur, especially if pieces of the uroliths were not completely removed.

Inguinal Hernia

After a male horse has been diagnosed with an inguinal hernia, treatment should commence immediately. In newborn foals, if the hernia is small and non-strangulated, the hernia can be manually reduced six times daily for two to four months. Typically, the hernia will then resolve in this time period, but will need to be carefully monitored to make sure the hernia contents (intestines) do not strangulate. In adult stallions or in male foals with strangulated hernias, surgery should be performed. Surgery consists of a bilateral castration with closure of the inguinal ring. If the intestines have been compromised, an intestinal resection along with an anastomosis should be performed. The prognosis for recovery from a strangulated inguinal hernia is guarded, as the survival rates can fall below 50%.

Urolithiasis

A horse can have uroliths (stones) in its urinary tract, a condition known as urolithiasis. The most common place for uroliths to be found in the horse is in the bladder. Most horse uroliths are composed of calcium carbonate. Uroliths in the bladder cause clinical signs such as bloody urine, dysuria, and pollakiuria. The horse may stretch its body when urinating and the skin around the urethra opening may become scalded with urine. The uroliths can also be a cause of colic, especially if they become trapped in the urethra. A horse with a blocked urethra due to a urolith will urinate frequently and have a distended bladder. Treatment is usually surgical in order to remove the urolith and treat any concurrent damage and infection caused by the stone.

Marine Mammals

Sea Lions

Pinnipeds can suffer from a number of ectoparasites. The ectoparasites that cause problems in pinnipeds are sucking lice and mites. Both pests cause alopecia in the pinniped. Lice (*Anoplura*) can cause debilitation and make the animal more susceptible to other infections. It is more common for sea lion pups to have this condition. Crowded conditions can exacerbate the problem.

Sea lions can also be infested with mites (*Demodex zalophi*) similar to demodex in dogs. Animals with mites have thickening of the skin due to hyperkeratosis. The mite infestation can be demonstrated by skin scrapings. Lice are treated with ivermectin, disophenol, dichlorvos, or topical rotenone. Mites can be treated with Amitraz or ivermectin.

Manatees

The manatee is an endangered animal in the USA with fewer than 1500 of the species left. Human encroachment on the manatee's habitat, along with boating collisions, is responsible for the low numbers. Venous blood is collected from the brachial area on the manatee's forelimb. Female manatees have a single mammary gland located under each flipper. The manatee calf will nurse from its mother for a minimum of 18 months. Captive manatees are fed a diet of monkey chow, carrots, cabbage, alfalfa cubes, lettuce, and bananas. They are also supplemented with calcium, phosphorus, and a multivitamin. Some health issues affecting manatees are cold stress, papilloma virus, and poisoning from red tides.

Sea Otters

The sea otter belongs to the family Mustelidae. They have very little body fat, no scent glands, and have loose skin. The male of the species can weigh over 100 pounds and can measure as long as five feet. Otters have extremely flexible spines and do not have clavicles. The thoracic cavity is very large and the animal has a large lung capacity to assist with buoyancy. The sexes are usually segregated into separate areas. Females may live with other adult females and their young. The female sea otter will have first estrus around four years of age. They are seasonally polyestrous. The male reaches sexual maturity around the age of five. The female will most typically have one pup.

Due to their limited habitat and off-shore location, the sea otter can become a victim of oil spills. Sea otters that are oil-covered need to be immediately treated. The oil-covered animal needs to be washed with liquid dish soap and water. Body temperature should be monitored during the washing and rinsing periods. Because of the danger of ingesting the oil and the damage the oil can cause, medical treatment is necessary. This consists of fluids (IV or subcutaneous), activated charcoal, steroids, and antibiotics. The animals should then be towel-dried or dried with a low-temperature hair drier. Ingestion of crude oil can cause liver necrosis, liver lipidosis, stomach erosions and hemorrhage, and renal damage.

Seals

A seal (Pinniped) is a large marine mammal with legs that act as flippers and no tail. Most seals eat fish and other sea food. There are three separate families of seals: true seals, eared seals, and walrus. The true seal (phocid) is distinguished by short flippers that are not adaptable to walking on the land; they also lack external ears. Examples of true seals include the harbor seal, harp seal, and elephant seal. The eared seal (otariid) has tiny ears and uses its flippers to lift its body off the ground. Examples of eared seals include sea lions or the furred seals. The walrus (odobenids) is the only member of the walrus family and is characterized by its tusk. All walruses are excellent swimmers and move on land by flexing their bodies.

Scombroid Fish Poisoning

Scombroid fish poisoning occurs when scombroid fish such as tuna and mackerel are not properly preserved. These fish have high levels of histamine and when they are poorly handled, can have toxic histamine levels. These fish must be used within a short time of being caught even if they are frozen. Animals that have consumed toxic fish (pinnipeds) show signs of depression, do not want to eat, have an inflamed throat and mouth, and have conjunctivitis. Some animals have abdominal pain, diarrhea, vomiting, or itching. Antihistamines provide some relief, but generally, the animal will recover within a few days provided the source fish is not continued to be fed.

Stranded Whales

Mass stranding of whales (cetaceans) is when two or more animals beach themselves. The cause of these mass strandings is uncertain. Whales may strand themselves because of biotoxins that contaminate the water or because of a lack of food. One theory as to why whales beach themselves is poor sonar reflection off of beaches that slope to the ocean. Another is that geomagnetic disturbances occur that cause the whales to misinterpret geomagnetic cues. The whales may strand themselves due to destruction of their acoustic system, or because of difficulty navigating unfamiliar coastlands or being in shallow water. A final theory is that underwater disturbances such as earthquakes or volcanic activity may cause the whale to beach itself.

Research

Animal Welfare Act

The federal Animal Welfare Act was passed to ensure that animals used in research, for exhibition, or as pets are provided humane care and treatment. It also is used to ensure the humane treatment of animals when transported. The owners of animals are protected from the theft of their animals by preventing

the sale or use of stolen animals. This act also regulates the purchase, transportation, sale, housing, care, treatment, and handling of animals used for research, for experiments, for exhibition, or intended for sale as pets. An example of a requirement from the federal Animal Welfare Act is that measures must be taken to ensure the psychological well- being of primates used in research.

Animals Used in Research

Mice

Mice are the most common animal model in research. They are used in many types of research: genetic testing, immunology, toxicology, cancer, metabolism, diabetes, obesity, cardiovascular studies, and aging. They are especially useful because of their short reproductive cycle. Mice are polyestrous, undergo spontaneous ovulation, and have a four- to five-day estrous cycle. The gestation period of mice is 19 to 21 days. Their average life span is two to three years and adults typically weigh between 20 and 40 grams. Mice can suffer from Tyzzer's disease caused by *Clostridium piliforme,* an obligate, intracellular, spore-forming bacterium. Clinical signs in the laboratory mouse vary because of the many different strains of laboratory mice. Some animals can be seemingly healthy carriers of the disease or some strains may have high mortality levels.

Ferrets

Ferrets (*Mustela putorius furo*) are induced ovulators that may develop pancytopenia as a result of high estrogen levels produced by mature follicles. This can be prevented by spaying the ferret. Common neoplasms of ferrets are insuloma, lymphosarcoma, chordoma, and adrenocortical adenoma/adenocarcinoma. Ferrets can become infected with the human influenza virus and are used as an animal model for human influenza research. Musk is a normal product of the ferret's sebaceous glands and is influenced by sex hormones. Removal of the gonads and scent glands reduce this odor production. The female ferret can become extremely aggressive when mating and may kill the male in her territory. In mating, always take the female to the male's cage and remove the female as soon as possible.

Rabbits

Rabbits are induced ovulators and the rabbit uterus contains two cervices. The female rabbit is polyestrous and can be bred at six to seven months of age. Parturition in rabbits is called kindling. Newborn bunnies are called leverets. Newborn rabbits typically nurse only once a day. Rabbits are often used to study respiratory diseases like asthma and cystic fibrosis (newborn rabbits). They are also used in testing for safety and used to produce antibodies.

Pasteurella multocida causes snuffles in rabbits. The disease causes upper respiratory signs such as a thick nasal discharge, conjunctivitis, and ear infections. Rabbit incisors grow constantly and must be worn down by chewing. Teeth that have malocclusion need to have regular cutting.

Guinea Pigs

Because guinea pigs are fully developed at birth (precocious), they are valuable in germ-free research. The female guinea pig ovulates spontaneously; mating is not necessary for ovulation to occur. Female guinea pigs should be bred before they pass seven months of age. Otherwise, their pelvic symphysis will fuse. The pregnant guinea pig may suffer from pregnancy toxemia. Factors that put a guinea pig at risk for pregnancy toxemia include obesity, being in the first or second pregnancy, heredity, or a change in diet. Vitamin C deficiency in the guinea pig produces the clinical signs of a rough haircoat, anorexia, dehydration, swollen joints, and gingival hemorrhage. *Bordetella bronchiseptica* is an important cause of respiratory disease in the guinea pig. Molar teeth in guinea pigs sometimes need to be floated, especially as they age.

Norwegian Rats

The laboratory rat is the Norwegian rat (*Rattus norvegicus*). These rats serve as models for many human disorders. They have an average life span of 2.5 to 3.5 years. As adults, the males weigh from 400 to 500 grams; female adults weigh 250 to 300 grams. Rats reach puberty at about 50 days of age. They should not be housed alone, as they are social animals. Some behaviors rats exhibit are wrestling, standing on their hind legs to explore their environment, and grooming. One curious anatomic anomaly is that a rat does not have a gallbladder. Sialodacryoadenitis is a rat disease caused by a coronavirus. This disease causes the following clinical signs: epiphora, swelling of the ventral neck, and megaglobus. Ringtail in rats is a disease caused by low humidity and high ambient temperature. The clinical signs are dry gangrene and annular constrictions of the tail.

New World Monkey

New World monkeys are used in animal research and are valued for their small size and low risk of zoonotic disease; however, they are not as commonly used as Old World primates. The commonly used New World monkeys in research include squirrel monkeys, marmosets, titi monkeys, and owl monkeys. Marmosets belong to the family Callitrichidae; the common marmoset (*Callithrix jacchus*) is used for laboratory research. Owl monkeys are nocturnal animals and belong to the genus *Aotus* . There are many types of squirrel monkeys used in research. This monkey is highly social and needs to be maintained in a social

group. The titi monkey (*Callicebus cupreus*) forms very strong mating bonds and is very sensitive to changes in the environment.

Vitamin C and D Deficiency

Vitamin C deficiency is a common problem in primates used for laboratory research. A lack of adequate vitamin C levels in the primate diet can lead to immunosuppression, an increase in contracting infections, and scurvy. Commercial primate diets typically have vitamin C added but the supplement only lasts for three months after the diet has been started. Primates should be supplemented with citrus fruits and green leafy vegetables to ensure that they are receiving proper levels of vitamin C. New World primates are susceptible to a deficiency in vitamin D and are unable to use the form of vitamin D found in plants (vitamin D2). They need to be supplemented with vitamin D3, which can be found in fish-liver oils. Vitamin D deficiency can lead to osteomalacia, osteodystrophia fibrosa, and rickets.

NAVLE Practice Test

1. An 8-year-old thoroughbred mare is evaluated for a 1-year history of infertility and atypical aggressive behavior. Physical exam findings are normal except for an enlarged ovary discovered on rectal palpation. Ultrasonography demonstrates a multiloculated appearance of the enlarged ovary. Which of the following assays, when elevated, is the most consistent indicator of the presumptive diagnosis?

a. Progesterone
b. Estrogen
c. Testosterone
d. FSH
e. Inhibin

2. A 4-year-old ferret presents with weakness, depression, lethargy, ptyalism, and posterior paresis. The owner indicates that these symptoms usually occur every morning before feeding and typically become less pronounced after the patient eats. As of late, the symptoms have become more severe and prolonged. Which of the following is the most likely diagnosis?

a. Adrenal cortical neoplasia
b. Insulinoma
c. Lymphoma
d. Leiomyosarcoma
e. Pancreatic exocrine adenocarcinoma

3. A distressed and vocalizing 7-year-old domestic shorthair cat is evaluated for acute bilateral hind-limb paralysis. On physical examination, a grade 3/6 left-sided systolic murmur is auscultated. The patient's femoral pulses are absent and the footpads are cold and pale. There is also painful contracture of the gastrocnemius and quadriceps muscles. Which of the following conditions is the most likely diagnosis?

a. Intervertebral disc disease
b. Fibrocartilaginous infarct
c. Arterial thromboembolism
d. Spinal cord neoplasia
e. Viral myelitis

4. Which of the following is the most common cause of exophthalmos in cattle?

a. Sinusitis
b. Extracranial meningioma
c. Retrobulbar lymphosarcoma

d. Glaucoma
e. Orbital arteriovenous fistula

5. An 8-year-old Labrador retriever is evaluated for progressive foreleg lameness of approximately 2 months' duration. On physical examination, there is marked soft tissue swelling involving the carpus and palpation of this area produces significant pain. Radiographs of the affected area demonstrate severe lysis of the distal radius with erosion of cortical bone. A needle biopsy displays osteoblasts with frequent mitotic figures, eccentric nuclei, and deeply basophilic cytoplasm. If amputation is elected by the owner, what is the most appropriate pharmacotherapy following the procedure?

a. Vincristine
b. Prednisone
c. Cyclophosphamide
d. Azathioprine
e. Cisplatin

6. A 2-year-old male German shepherd is evaluated for marked weight loss, listlessness, and frequent, voluminous stools that have developed over the past few months. The owner states that the patient is eating more than usual, even his own stools. Physical examination yields a body condition score of 2/5, poor hair coat, dull mentation, and generalized ill thrift. Steatorrhea is noted when the patient defecates on the floor. Which of the following tests is most appropriate to confirm the presumptive diagnosis?

a. Intestinal biopsy
b. Glucose curve
c. Ultrasonography
d. Trypsin-like immunoreactivity
e. Fecal flotation

7. What is the most common cause of egg binding and dystocia in cage birds?

a. Hypocalcemia
b. Obesity
c. Oviduct damage
d. Sedentary lifestyle
e. Genetic predisposition

8. A 13-year-old female domestic shorthair cat is discharged from a clinic following bilateral thyroidectomy. Two days later, the patient is reevaluated for panting, facial rubbing, restlessness, and one seizure that occurred an hour ago. What postsurgical complication is the most likely explanation for these clinical signs?

a. Horner's syndrome
b. Laryngeal paralysis
c. Renal disease
d. Hypocalcemia
e. Hypoparathyroidism

9. After being hit by a car, a dog presents with a non-weight-bearing lameness of the foreleg. On physical exam, there are no palpable fractures. The limb itself just hangs without any movement or any pain sensation. The dog is also found to have a Horner's syndrome. What is the most likely diagnosis that would explain these exam findings?

a. Cervical disc disease
b. Arterial thromboembolism
c. Fibrocartilaginous infarct
d. Radial nerve paralysis
e. Brachial plexus avulsion

10. The most common cause of bony facial masses in llamas is

a. Osteomyelitis
b. Tooth root abscess
c. Ossifying fibroma
d. Facial bone fracture
e. Ameloblastoma

11. A 5-year-old corgi presents with opisthotonus, thoracic limb hyperextension, and rear limb paraplegia following a fall from a 25-foot cliff. Which of the following is the most likely location of the lesion?

a. C1-C5
b. C6-T2
c. T3-L3
d. L4-S3
e. S1-S3

12. Which of the following is the most common organism found in feline pyometra?

a. Staphylococcus aureus
b. Pasteurella multocida
c. Streptococcus pyogenes
d. Escherichia coli
e. Pseudomonas aeruginosa

13. A 19-year-old male fjord pony is evaluated for a 3-day history of moderate to severe bouts of left-sided epistaxis. On presentation, the pony is quiet, alert, and

responsive. The heart rate is 60, respiratory rate is 40, and rectal temperature was 101°F. The hematocrit is 31% and total protein is 6.5 g/dL. There is no evidence of trauma or swelling over the sinus region of the head, and airflow through each nostril is not particularly impeded. Which of the following diagnostic tests will provide the most useful information to confirm a presumptive diagnosis?

a. Thoracic ultrasonography
b. Endoscopic examination
c. Transtracheal aspiration
d. Head/Thorax radiography
e. Bronchoalveolar lavage

14. A 5-year-old jersey cow is evaluated for an abnormal pelvic gait that started in the right hind leg a month ago, but as of 3 days ago, is now present in both limbs. The dairy farmer mentions that she mated with a bull last month. Upon observation, the cow is able to move her pelvic limbs forward, but does so with an overflexed (dropped) tarsus and dorsal buckling of the metatarsophalangeal joint. Pain perception was normal on all surfaces of the pelvic limbs. Tail and anal function/tone were also normal. The dysfunction of which of the following nerves is responsible for these physical exam findings?

a. Peroneal nerve
b. Sciatic nerve
c. Tibial nerve
d. Obturator nerve
e. Femoral nerve

15. A 3–month-old quarter horse foal is evaluated for an acute onset of tachypnea, inappetence, and a mild mucopurulent nasal discharge. Physical examination yields a temperature of 104.5°F, a respiratory rate of 60, and wheezes and crackles on thoracic auscultation. Thoracic radiographs reveal a diffuse interstitial pattern with areas of abscessation. Which of the following is the most likely diagnosis?

a. Rhodococcus equi pneumonia
b. Undifferentiated bronchopneumonia
c. Equine herpesvirus I
d. Parascaris equorum infection
e. Bronchointerstitial pneumonia

16. What is the most common cause of facial paralysis and a head tilt in a young calf?

a. Meningoencephalitis
b. Otitis media

c. Trauma
d. Tick paralysis
e. Botulism

17. Which of the following syndromes is the most common cause of feline stomatitis?

a. FeLV/FIV infection
b. Neoplasia
c. Chronic renal failure
d. Periodontal disease
e. Toxin ingestion

18. A 3-month-old Yorkshire terrier presents with a history of episodic seizures, ataxia, weakness, and vomiting. Aside from the patient's relatively small size (owner indicates the patient was the runt of the litter), physical examination does not yield any significant findings. Initial diagnostics include comprehensive blood work, which reveals a microcytosis of the RBCs as well as a decrease in creatinine, BUN, and glucose. Radiographic findings demonstrate a mild microhepatica. Which of the following tests is the next step to help rule out or support the presumptive diagnosis?

a. Radiographic portography
b. Liver biopsy
c. Serum bile acids
d. Colorectal scintigraphy
e. Doppler ultrasound

19. What is the most common cause of acquired megaesophagus in the dog?

a. Pyloric stenosis
b. Polyradiculoneuritis
c. Esophageal foreign body
d. Myasthenia gravis
e. Lead toxicity

20. A 6-year-old male domestic shorthair cat is evaluated for vomiting, ataxia, depression, and anorexia that started this morning, shortly after the patient returned after having been gone for 2 days. Initial blood work demonstrates an elevated BUN and creatinine, as well as an increased anion gap of 45 mEq/L. Microscopic examination of urine sediments reveals calcium oxalate crystals. Which of the following is the most appropriate initial treatment for the presumptive diagnosis?

a. 4-methylpyrazole
b. Ethanol

c. Hemodialysis
d. Emesis induction/activated charcoal
e. Aggressive fluid therapy

21. A 10-year-old neutered male domestic shorthair cat, which was diagnosed with diabetes mellitus 8 weeks ago, presents for an evaluation of glycemic control. The patient is currently on 2 units of PZI insulin twice daily, and according to the owner was doing great during the initial 7 weeks of treatment. The patient gained some weight and was no longer demonstrating polyuria or polydipsia. During this last week, however, the patient had 3 seizures as well as a few periods of inappetence. The owner did not change the insulin dose and properly stores the medicine in the refrigerator. Physical exam findings are uneventful. The patient is BAR and appears quite content. Initial blood work reveals a glucose of 95 mg/dL and a fructosamine level of less than 250 micromol/L. Which of the following is the most likely explanation for these historical and diagnostic findings?

a. Somogyi phenomenon
b. Pancreatitis
c. Hypocortisolism
d. Transient diabetes mellitus
e. Insulinoma

22. An owner that develops a pruritic rash on his arms shortly after acquiring a 16-week-old Vietnamese potbellied piglet most likely has:

a. Demodex phyllodes
b. Haematopinus suis
c. Sarcoptes scabei
d. Microsporum nanum
e. Amblyomma naponense

23. Which of the following is the definitive host for Taenia saginata?

a. Dogs
b. Mice
c. Cattle
d. Cats
e. Humans

24. A 5-year-old neutered male Basset Hound is evaluated for an acute onset of inappetence and ocular pain in the left eye. The owner states that the patient has been hiding under the table and rubbing his eye on the carpet. Ocular examination reveals blepharospasm, corneal edema, hyperemia, scleral injection, and an inconsistent menace response. Fluorescein stain test is

negative, as is the Schirmer tear test. A Tono-Pen yields an average intraocular pressure of 45 mm/Hg. Which of the following medications is contraindicated in this case?

a. Mannitol
b. Acetazolamide
c. Latanoprost
d. Atropine
e. Oral glycerin 50%

25. A 6-year-old neutered male domestic shorthair cat is evaluated for an acute episode of respiratory distress. In the exam room, the patient is open-mouth breathing and exhibiting an abdominal push on expiration. Physical exam findings include a temperature of 101°F, a respiratory rate of 30, and a heart rate of 180. There are increased bronchovesicular sounds on auscultation. Heart sounds are normal. Initial blood work is within normal limits. Radiographs demonstrate aerophagia and hyperinflation of the lung fields as well as diffuse bronchial wall thickening. Which of the following is the most likely diagnosis?

a. Hypertrophic cardiomyopathy
b. Feline asthma
c. Pneumonia
d. Primary or metastatic neoplasia
e. Paragonimus kellicotti infection

26. In canines and felines, what is the most common cause of toxin-induced seizures?

a. Metaldehyde
b. Pseudoephedrine
c. Organophosphates
d. Caffeine
e. Ethylene glycol

27. A 5-year-old lactating Holstein cow is evaluated for acute anorexia and decreased milk production for 24 hours. She is currently being treated for mastitis and was responding favorably to the treatment up until today. Physical examination demonstrates a normal temperature, heart rate, and respiratory rate. A ping is auscultated on simultaneous percussion and auscultation of the left side of the abdomen between the 9th and 13th ribs dorsally. Which of the following laboratory findings would support the presumptive diagnosis?

a. Hyperchloremia
b. Hypokalemia
c. Hypercalcemia

d. Hyperkalemia
e. Hypernatremia

28. A 7-year-old female miniature dachshund is evaluated for an acute onset of vomiting and profuse, bloody diarrhea. Diet has remained unchanged and the patient is vaccinated regularly at the clinic. Initial diagnostics reveals a hematocrit of 65% without a leukopenia, and a fecal cytology displays increased numbers of clostridial organisms. The patient is very tender during abdominal palpation, but only thickened loops of bowel are found. Which of the following syndromes is the most likely diagnosis?

a. Pancreatitis
b. Inflammatory bowel disease
c. Intussusception
d. Parvovirus
e. Hemorrhagic gastroenteritis

29. Which of the following bowel segments is the most common location for an intussusception in the canine?

a. Duodenum
b. Jejunum
c. Ileocecocolic junction
d. Ascending colon
e. Pylorus

30. Which of the following drugs is most widely used to prevent exercise-induced pulmonary hemorrhage (EIPH) in racehorses?

a. Dexamethasone
b. Furosemide
c. Terbutaline
d. Aminophylline
e. Epinephrine

31. An 11-year-old spayed female Collie is evaluated for a 2-month history of hematuria, stranguria, and pollakiuria that has not resolved with antibiotic therapy. Physical exam findings are within normal limits. A technician collects urine via a cystocentesis for urine cytology. Microscopic examination demonstrates large, round epithelial cells that exhibit macronucleoli, nuclear molding, and occasional multinucleated cells. A soft tissue opacity and numerous blood clots are found in the bladder trigone on ultrasonographic examination. With or without surgery, which of the following therapeutic regimes are most commonly employed to medically manage the presumptive diagnosis?

a. Radiation
b. Systemic chemotherapy with cisplatin
c. Intravesical doxorubicin
d. Intravesical hyperthermia with cisplatin in warmed fluids
e. Piroxicam and misoprostol

32. Which of the following conditions exhibits a reverse "C" sign on a canine radiograph?

a. Mesenteric torsion
b. Gastric dilatation-volvulus
c. Intestinal volvulus
d. Ileocecocolic intussusception
e. Splenic torsion

33. A 3-month-old stray kitten is evaluated for lethargy, anorexia, and ptyalism. Physical exam findings include a temperature of 105°F, heart rate of 180, and respiratory rate of 15. There is a mild serous ocular and nasal discharge as well as ulcers of the tongue and hard palate. Which of the following pathogens is the most likely source of infection?

a. Feline calicivirus
b. Feline leukemia virus
c. Feline Chlamydia psittaci
d. Feline herpes virus
e. Mycoplasma felis

34. Which of the following injections is necessary as a supplement in 3- to 5-day-old piglets?

a. Calcium gluconate
b. Magnesium sulfate
c. Zinc sulfate
d. Ferrous sulfate
e. Potassium phosphate

35. Which of the following conditions in cattle is NOT a reportable disease?

a. Scabies
b. Vesicular stomatitis
c. Brucellosis
d. Infectious bovine rhinotracheitis
e. Screwworm

36. An 8-year-old spayed female cat is evaluated for inappetence, lethargy, and open-mouthed breathing. The owner states that this has been going on for a week and is becoming progressively worse. It is very difficult to conduct a

physical examination on the patient since she becomes easily stressed with a subsequent cyanosis of the mucous membranes. A brief auscultation is achieved, which demonstrates harsh lung sounds and a pronounced systolic murmur over the left side of the thorax. Further diagnostic cannot be performed without oxygen treatment, to allow the patient to calm down, followed by sedation to eliminate the stress associated with more testing. Which of the following anesthetic agents is most appropriate in this scenario?

a. Ketamine
b. Thiopental
c. Etomidate
d. Propofol
e. Dexdomitor (dexmedetomidine)

37. Which of the following conditions is the most common cause of feline obstipation?

a. Nerve injury
b. Colonic neoplasia
c. Manx spinal cord deformity
d. Idiopathic megacolon
e. Pelvic canal stenosis

38. A 2-year-old male thoroughbred is evaluated for exercise intolerance and an upper respiratory tract noise, described as a "roaring" by the trainer, which occurs during strenuous exercise. Physical examination of the resting horse is within normal limits except for a palpable prominence over the left muscular process of the arytenoid cartilage. The horse is exercised for 15 minutes to elicit the respiratory noise that's origin is respiratory. The horse has no trouble eating or drinking and has no nasal discharge or coughing. Which of the following is the most likely diagnosis?

a. Dorsal displacement of the soft palate
b. Pharyngeal lymphoid hyperplasia
c. Pharyngeal cysts
d. Epiglottic entrapment
e. Laryngeal hemiplegia

39. A 2-year-old female cockatiel is evaluated for inappetence, polyuria, vomiting, and lethargy over the past 24 hours. The owner lets him out of the cage while he is home; however, the patient is not always supervised. Physical examination is brief to prevent stress and subsequent collapse of the patient, but no overt abnormalities are noted. The patient is placed in an oxygen cage for 30 minutes prior to radiology, which demonstrates a large amount of dense, radiopaque material in the GI tract. The owner mentions that he has recently

caught the patient chewing the paint off the wall adjacent to its cage. Which of the following is the most likely diagnosis?

a. Lead poisoning
b. Chlamydiosis
c. Proventricular dilatation disease
d. Pancreatitis
e. Diabetes mellitus

40. A 13-year-old Arabian stallion is evaluated for a unilateral enlargement of the scrotum. The enlargement has been progressive during the preceding 6 months, but dramatically so during the last 3 weeks. There is no history of trauma. The owner also states that the stallion has had less successful breeding over the last 8 months. Physical examination demonstrates a unilaterally enlarged scrotum that is not painful or hot on palpation. Ultrasonography of the affected testicle exhibits randomly distributed hyperechoic regions. Which of the following conditions is the most likely diagnosis?

a. Epididymitis
b. Torsion of the spermatic cord
c. Orchitis
d. Hematocele
e. Seminoma

41. An 8-year-old spayed female Labrador is evaluated for urinary incontinence. The owner reports that the problem occurs only at night when the patient sleeps. During the day she voids normally at her usual times. She is not receiving any medications nor does she have any concurrent medical condition or past trauma. Physical examination results are within normal limits. The patient is taken outside to void for a free catch urine sample. The urine stream and volume are normal as are the results of the urinalysis. Radiographic findings yield no significant results. The veterinarian makes a presumptive diagnosis of decreased urethral tone and suggests the owners try medical management for a few months followed by a scheduled recheck. Which of the following drugs is most appropriate to manage this patient?

a. Phenoxybenzamine
b. Dantrolene
c. Phenylpropanolamine
d. Bethanechol
e. Propantheline

42. A 3-year-old Appaloosa gelding is evaluated for an acutely inflamed right eye. The owner indicates that the horse was fine this morning and that nothing traumatic happened in the interim. Physical examination findings, not including

the eye, are normal. On examination with an ophthalmoscope, the iris appears dull and swollen, and the pupil is constricted. The anterior chamber has a cloudy appearance as does the cornea. If this condition is not treated promptly and properly, what is likely to develop?

a. Lens luxation
b. Retinitis
c. Posterior synechia
d. Corneal ulcer
e. Equine night blindness

43. A 3-month-old male Hereford calf is evaluated for drooling, inappetence, and labored breathing, which the rancher first noticed this morning. No other calves are affected at this time. Physical exam reveals a fever of 106°F and an inspiratory stridor on thoracic auscultation. Oral examination exposes a severely swollen and inflamed tongue as well as an extremely ulcerated buccal mucosa and soft palate that are particularly foul smelling. They larynx is swollen on palpation as well. Which of the following is the most likely diagnosis in this case?

a. Infectious bovine rhinotracheitis
b. Necrotic laryngitis
c. Pharyngeal trauma
d. Actinobacillosis infection
e. Vesicular stomatitis

44. Which of the following infectious agents is responsible for necrotizing hemorrhagic enteritis in newborn foals?

a. Clostridium perfringens
b. Cryptosporidium parvum
c. Strongyloides westeri
d. Rotavirus
e. Escherichia coli

45. A sternally recumbent 6-year-old female Holstein cow is evaluated for depression, anorexia, and muscle tremors over the past 6 hours. According to the farmer, the patient calved 3 days ago and has no history of postpartum illness. Physical exam reveals shallow respiration, ear twitching, decreased intensity of heart sounds, and an absence of gut sounds over the rumen. Which of the following is the most likely diagnosis?

a. Toxic metritis
b. Calving paralysis syndrome
c. Pelvic fracture

d. Compartment syndrome
e. Postparturient paresis

46. A 3-day-old male Arabian foal is evaluated for progressive lethargy, inappetence, and abdominal swelling over the past 24 hours. The owner reports that the foal was born normally and nursed within 20 minutes. Physical examination reveals tachycardia, tachypnea, and a fluid wave produced with abdominal ballottement. During the examination the foal urinates a small amount, and then voids more 15 minutes later. Serum chemistry values indicate hyperkalemia, hyponatremia, and azotemia. An abdominocentesis is performed and the fluid submitted for analysis. The concentration of potassium and creatinine in the collected fluid is twice that of the levels found on the serum chemistry. What type of fluid was recovered from the abdominocentesis?

a. Bile
b. Urine
c. Chyle
d. Pus
e. Blood

47. Which of the following is the most appropriate treatment for poultry coccidiosis?

a. Tetracycline
b. Amprolium
c. Nystatin
d. Penicillin
e. Neomycin

48. Which of the following is the treatment of choice for Eperythrozoonosis suis in swine?

a. Tetracycline
b. Erythromycin
c. Penicillin
d. Bacitracin
e. Sulfadimethoxine

49. An 8-week-old kitten recently adopted from an animal shelter presents with depression, anorexia, and a purulent nasal and ocular discharge. Physical exam reveals a temperature of 103.3 and a severe bilateral conjunctivitis with a concurrent ulcerative keratitis that is discovered upon closer examination with an ophthalmoscope and fluorescein stain. Which of the following pathogens is most likely responsible for these clinical signs?

a. Feline leukemia virus
b. Feline calicivirus
c. Mycoplasma felis
d. Feline herpesvirus
e. Chlamydia psittaci

50. Which of the following is the appropriate treatment for nitrate toxicosis in ruminants?

a. Acetic acid
b. Methylene blue
c. Penicillamine
d. Acetylcysteine
e. Calcium disodium edetate

Answer Key and Explanations

1. E: Inhibin. The presumptive diagnosis in this case is granulosa cell tumor (GCT), which represents the most common neoplasia of the reproductive tract in mares. Mares with granulosa cell tumors may have elevated serum concentrations of estrogen, testosterone, and/or inhibin; however, inhibin is the hormone that is most consistently and reliably increased in affected mares, and is therefore a more reliable indicator of the disease. Inhibin is a hormone that may be produced by granulosa cell tumors and acts to suppress follicle-stimulating hormone (FSH) secretion by the anterior pituitary. As a result of this negative feedback, follicular growth of the contralateral ovary is severely decreased, with a resultant decrease in size.

2. B: Insulinoma. Insulinomas are the most common tumors in ferrets between 3 and 5 years of age. They are insulin-secreting beta-cell tumors that produce insulin, and thus, create clinical signs associated with hypoglycemia (ie. depression, collapse, seizures, lethargy, ptyalism, and paresis). The symptoms may be exacerbated following periods of glucose consumption or depletion, as seen with exercise or overnight fasting. Diagnosis is a blood glucose level of less than 60 mg/dL and an insulin level of greater than 250 pmol/L.

3. C: Arterial thromboembolism. Thrombus formation within the left heart with resultant systemic embolization at the aortic trifurcation is a serious and common sequela of myocardial disease in the cat. Clot formation within the left heart is ameliorated by decreased blood flow and endothelial damage associated with cardiac disease. When an embolus becomes loose and is transported into the systemic circulation, it will commonly occlude the distal aortic trifurcation and produce symptoms such as intense vocalization (pain), paralysis or paresis, absent femoral pulses, and cold extremities. Medical management involves treating the underlying heart disease, providing exercise restriction, and administering low-dose aspirin every 3 days.

4. C: Retrobulbar lymphosarcoma. Lymphosarcoma that invades the retrobulbar tissues is the most common cause of exophthalmos in cattle and usually carries an extremely poor prognosis because affected cattle usually live less than a year after diagnosis. Other physical exam findings may include lymphadenopathy or melena due to diffuse spread of the neoplasia.

5. E: Cisplatin. Based on the history and diagnostic findings of this patient, the presumptive diagnosis is osteosarcoma of the distal radius, which represents the most common bone neoplasia in large breed dogs. Once diagnosed, the treatment of choice is amputation of the affected limb followed by

chemotherapy with the drug cisplatin, which usually affords a survival rate of little over a year.

6. D: Trypsin-like immunoreactivity. Exocrine pancreatic insufficiency (EPI) is the ailment in this case scenario and is seen commonly in young German Shepherds. It occurs when the exocrine pancreas produces inadequate amounts of enzymes for digestion, leading to maldigestion of the intestinal contents. The test of choice for EPI is trypsin-like immunoreactivity, which measures specific pancreatic enzymes (trypsin) in the bloodstream. In EPI, these enzymes are not being produced in sufficient amounts, so the TLI results are profoundly decreased.

7. A: Hypocalcemia. Calcium malnutrition or abnormal calcium metabolism are frequent causes of egg binding and dystocia in cage birds. Calcium is not only necessary for normal egg development, but also for smooth muscle contractility that aids in moving the egg along the reproductive tract and out the cloaca. High-fat diets, such as seed exclusive diets, are likely culprits in most cases because they lack calcium as well as several minerals that are necessary for normal reproductive function. Certain cage birds, such as budgerigars and cockatiels, are prone to higher mortality when they present with hypocalcemia and dystocia, and so must be handled with great care. Fluid therapy, parenteral calcium, oxytocin, and the last resort, surgery, are the common treatment options.

8. D: Hypoparathyroidism. Hypoparathyroidism, which leads to hypocalcemia, is the most important, potentially fatal complication of bilateral thyroidectomy. It occurs as a result of damage to or accidental removal of the parathyroid gland during the surgery. Most animals do not demonstrate clinical signs until serum calcium levels decrease to below 7.5 mg/dL, which usually occurs between 2 and 4 days following surgery. At this point, patients will begin to display symptoms such as seizures, inappetence, ataxia, lethargy, and restlessness.

9. E: Brachial plexus avulsion. The ventral branches of the C6-8 and T1-2 spinal nerves comprise the brachial plexus. When the thoracic limb is severely abducted from the body, usually as a consequence of extreme trauma, the spinal nerve roots become severely stretched or torn within the spinal canal, which leads to complete or partial paralysis of the affected limb. Damage to these nerve roots in the spinal cord also disrupts sympathetic innervation to the eye (same side as affected forelimb), which produces a Horner syndrome (miotic pupil, enophthalmos, ptosis, and protrusion of the third eyelid).

10. B: Tooth root abscess. Tooth root abscesses are the most common cause of facial swelling in llamas. They usually involve PM1, M1 and M2 of the mandible,

and, less commonly, the canines, incisors, and the maxillary teeth. This problem manifests as a growing swelling that drains pus intermittently. Radiographs are needed to confirm the source of the swelling. Treatment involves removal of the infected teeth and curettage of the empty socket.

11. C: T3-L3. The posture demonstrated by this patient, also known as Schiff-Sherrington syndrome, indicates a severe spinal cord lesion somewhere from T3 through L3. The mechanism by which this occurs involves inhibitory neurons called border cells that are located in the lumbar spinal cord (L1-L7). The axons of these neurons extend cranially to the cervical intumescence (C6-T2) where they provide tonic inhibition of the extensor muscle motor neurons in the cervical intumescence. If a lesion is present cranial to the border cells, and caudal to the cervical intumescence, tonic inhibition to the thoracic limbs via border cell axons is blocked, resulting in forelimb hyperextension.

12. D: Escherichia coli. Pyometra develops as a result of both repeated uterine exposure to progesterone and subsequent infection by opportunistic bacteria that ascend from the vagina (E.coli). Under the influence of progesterone, the endometrial glands of the uterus develop and produce large amounts of fluids and secretions, which create an excellent environment for bacterial growth and subsequent infection.

13. B: Endoscopic examination. Endoscopic examination of the ethmoids, guttural pouches, sinuses, and trachea will provide the most useful information regarding the cause of epistaxis in this patient. During endoscopy, the upper respiratory tract can be evaluated for the source of the bleeding and/or the presence of fungal plaques, tumors, ulcerative lesions, and ethmoid hematomas. Once the etiology is determined, the other tests mentioned above may be indicated to further evaluate the condition that is the cause of the epistaxis. For example, if an ethmoid hematoma is present, radiographs will confirm if the hematoma has expanded into the adjacent paranasal sinuses.

14. C: Tibial nerve. The most common injury that produces these clinical signs in an adult cow is fracture of the sacrum or caudal vertebrae, as a result of being mounted by a bull, with subsequent trauma to the tibial nerve. Depending on which vertebrae are injured, the signs may be isolated to the pelvic limbs, or, more commonly, include the tail, anus, and urethra, which result in urinary and/or fecal incontinence. In this patient, only the first 2 sacral nerves were affected, which produced a tibial nerve dysfunction. The tibial nerve innervates the extensor muscles of the pelvic limb. When this nerve does not function properly, the flexor muscles will take over and overflex the tarsus.

15. A: Rhodococcus equi pneumonia. R. equi pneumonia affects young foals between 3 and 6 months of age. Physical exam findings include tachypnea, fever, wheezes/crackles on auscultation, coughing, and mild nasal discharge. Blood work usually demonstrates a blood neutrophilia between 14,000 and 30,000/mcL, as well as a hyperfibrinogenemia. An interstitial lung pattern is normally seen on thoracic radiography. Bacterial culture of the organism will confirm the diagnosis.

16. B: Otitis media. Otitis media is the most common cause of facial paralysis and associated head tilt in a young calf. Any inflammation of the middle ear will have a direct effect on the facial nerve since they reside in close proximity to one another and are separated only by a thin layer of connective tissue.

17. D: Periodontal disease. The most common cause of feline stomatitis is periodontal disease. These cats are initially evaluated for dysphagia, inappetence, weight loss, ill thrift, pseudoptyalism and sometimes facial rubbing. Vocalization due to pain, especially during eating, is also common. Oral exam will demonstrate a bright red buccal mucosa, which can also affect the glossopalatine arch (faucitis), pharyngeal area (pharyngitis), or gingiva (gingivitis). Blood work is performed initially to rule out any underlying disease. Treatment involves a comprehensive dental to include scaling, polishing, and extracting of any teeth that show evidence of periodontal disease or odontoclastic resorptive lesions, a 4- to 6-week course of antibiotics as well as antiinflammatories, which may need to be continued indefinitely. A total tooth extraction may ultimately be necessary if a patient is refractory to treatment or medication.

18. C: Serum bile acids. Increased fasting or postprandial serum bile acid values help support a diagnosis of portosystemic shunt, which is the presumptive diagnosis in this case. Bile acids are substances that aid digestion. They are produced by the liver, stored in the gallbladder, and released into the intestinal tract where they are then reabsorbed in the ileum and returned via portal circulation to the liver for recycling. Portosystemic shunts are portal vessels that bypass the liver altogether, and thus never deliver the bile acids for recycling. As a result, bile acids will therefore remain in the systemic circulation in abnormally high concentrations.

19. D: Myasthenia gravis. Myasthenia gravis is the most common cause of megaesophagus in the dog. Acquired myasthenia gravis is an autoimmune disorder in which antibodies attack nicotinic acetylcholine (ACh) receptors, thus preventing the normal neuromuscular transmission that is needed for proper muscle function. Dogs with this disease may experience a generalized muscle weakness, or a more focal megaesophagus that can be visualized on survey

radiography. Diagnosis is made through a blood test that measures antibodies titers to the ACh receptors.

20. D: Emesis induction followed by activated charcoal administration. Treatment of ethylene glycol (EG) toxicosis should be immediate and is most successful if performed within 12 hours of ingestion. Since it is not known when exactly the patient consumed the antifreeze, it is still best to decontaminate first (unless the animal is comatose) by means of emesis and activated charcoal to reduce absorption of any EG that may still be in the GI tract. Fluid therapy (to correct dehydration and promote excretion of EG and its metabolites) and ethanol (prevents EG breakdown into toxic metabolites) are the next steps in treatment. 4-methylpyrazole is not recommended in cats only because the drug is cleared too quickly to be of any benefit.

21. D: Transient diabetes mellitus. A small percentage of diabetic cats (approximately 20%) are transiently diabetic and experience a remission of clinical signs within the first 8 weeks of treatment, which is why they must be closely monitored during the first few months. Insulin given to a patient in remission will create periods of severe hypoglycemia with resultant clinical signs such as seizures, ataxia, weakness, and anorexia. The glucose (95 mg/dL) reflects a low normal value. Fructosamine is also decreased at less than 250 micromol. Fructosamine is an excellent test to determine the patient's average blood glucose concentration over the previous 1 to 2 weeks. A level of less than 250 micromol is markedly decreased, which indicates that this patient experienced several episodes of hypoglycemia prior to evaluation. In this case, the insulin should be discontinued and the glucose closely monitored for at least 2 weeks.

22. C: Sarcoptes scabei. Sarcoptic mange is a common zoonotic ectoparasite in young pigs and is transmitted through general handling of these animals. The condition is usually transient and only lasts 7 days, but it does produce a large amount of discomfort and pruritus for both the owner and pig during this time. Affected pigs should be treated with ivermectin at weekly intervals for 4 weeks, which also serves to eliminate some of the common internal parasites as well. Affected owners should seek the advice of a dermatologist.

23. E: Humans. Humans are the definitive host for the tapeworm Taenia saginata, and become infected by ingesting undercooked beef that is infected with the intermediate form of the parasite called the cysticercus. Cattle become infected when they are grazing on land contaminated by human feces that contain the ova of the tapeworm.

24. D: Atropine. The patient described in this case is experiencing acute glaucoma. Initial emergency treatment is aimed at reducing the intraocular pressure as soon as possible to prevent optic nerve damage. Drugs that serve this purpose include mannitol, acetazolamide, pilocarpine, and oral glycerin 50%. Atropine, however, is contraindicated in glaucoma because it inadvertently increases intraocular pressure. Atropine dilates the pupil, which drives the iris into the angle of the anterior chamber, thus blocking the drainage of the aqueous humor.

25. B: Feline asthma. Feline asthma, also known as allergic airway disease, is an important respiratory condition in cats. It is characterized by bronchoconstriction of the small airways with resultant increased expiratory effort. In severe cases, many felines are open-mouth breathing and in obvious distress. In mild cases, cats may exhibit intermittent coughing or gagging. Radiographs normally reveal hyperinflation of the lung fields, aerophagia, and/or a peribronchial pulmonary pattern; however, they can sometimes appear quite normal. Treatment involves corticosteroids to reduce the airway inflammation, as well as bronchodilators to alleviate the airway constriction. Oxygen therapy is sometimes needed in severe cases.

26. C: Organophosphates. Insecticides, which include organophosphates and carbamates, are by far the most common cause of toxin-induced seizures in dogs and cats. These ingredients are frequently found in flea and tick control products such as shampoos, dips, and collars. Since they are so readily available for individual purchase in the pet stores, it is not uncommon for pets to be exposed to or overdosed with these products. Clinical signs include seizures, vomiting, hypersalivation, ataxia, depression, and, in severe cases, even death. Treatment involves evacuation of stomach contents (if a large amount is consumed), oxygen therapy, activated charcoal, hospitalization, and intravenous fluid therapy.

27. B: Hypokalemia. Left abomasal displacement is a common GI disorder of high-producing lactating dairy cattle. It is defined as a displacement of the abomasal body to the left dorsal abdominal area between the left body wall and the rumen. When this occurs, abomasal outflow is inhibited with a subsequent buildup of potassium, chloride, and hydrogen ions within the lumen of the abomasum. Because they are trapped, these ions become decreased in systemic circulation, thereby producing a metabolic alkalosis (hypochloremia and hypokalemia). Other factors that exacerbate this hypokalemia include anorexia, diarrhea, and vomiting.

28. E: Hemorrhagic gastroenteritis. A patient that presents with severe, acute bloody diarrhea and vomiting coupled with marked increase in packed cell

volume (PCV) (more than 50%), and with no concurrent leukopenia, is most likely suffering from hemorrhagic gastroenteritis. Increased numbers of clostridial organisms are usually found in a fecal smear, which are believed to be the source of the illness. Aggressive fluid therapy (± plasma transfusion), antibiotics, and antiemetics are required to successfully treat this disease.

29. C: Ileocolic junction. Intussusception usually occurs in very young animals (younger than 1 year) at the ileocecocolic junction. It is defined as the "telescoping" of one part of the intestine into an adjacent segment and yields clinical signs similar to those of a foreign body obstruction. There is usually a palpable cylindrical mass that is mildly painful on abdominal palpation. A diagnosis of intussusception is usually made from survey and contrast radiography as well as ultrasonography. Surgery is corrective.

30. B: Furosemide. The only medication that is permissible to prevent EIPH on race day is furosemide. EIPH is a common condition among racehorses following intense training or a race, which is characterized by an accumulation of blood and fluid in the airways due to an increase in pulmonary arterial blood pressure with resultant alveolar capillary rupture. Furosemide helps to reduce or prevent EIPH when given prior to a performance event (it is speculated) by reducing body water and intravascular fluid volume and thereby reducing the increased pulmonary arterial BP that accompanies exercise.

31. E: Piroxicam and misoprostol. Piroxicam is a nonsteroidal anti-inflammatory drug that is used to treat the presumptive diagnosis, a transitional cell carcinoma (TCC) of the bladder trigone. These tumors usually affect female dogs that are 10 years of age and older and cause a variety of clinical signs all related to urination (eg. stranguria, pollakiuria, dysuria, and hematuria). This rule out should be suspected in any older animal that presents with these signs despite previous antibiotic therapy. Diagnosis is made with urine cytology, ultrasound, and/or a double-contrast cystogram. Commonly, TCCs are nonresectable because they are located in the bladder trigone, and so medical management is sometimes the only option. Piroxicam inhibits tumor growth and, therefore, has the potential to improve the patient's quality of life or, in other instances, cause a complete remission altogether. Piroxicam frequently causes GI irritation, so concurrent use of misoprostol is often warranted.

32. B: Gastric dilatation-volvulus. A patient suffering from a gastric dilatation-volvulus will have a reverse C sign, or double bubble, when radiographed in right lateral recumbency. The reverse C sign refers to the appearance of the gas-filled pylorus and stomach when they rotate clockwise between 90° and 270°. When this occurs, the duodenum and pylorus move ventrally and to the left side and become lodged between the stomach and esophagus. A right lateral

radiograph will show the gas-filled pylorus situated cranially to the stomach and separated from the stomach by line of soft tissue.

33. A: Feline Calicivirus. A feline upper respiratory tract disease that produces ulcers of the tongue and hard palate and a significant fever is usually the result of a calicivirus infection. These viral infections can persist from 1 to 2 weeks and are commonly seen in cats with no previous or an unknown vaccine history. The virus is transmitted via cat-to-cat contact, airborne exposure, or through fomites. Cats are the source of infection and are either actively infected or asymptomatic carriers. Immunized cats are protected against systemic and severe respiratory disease, but could still develop a carrier state.

34. D: Ferrous sulfate. Piglets require supplemental iron (ferrous sulfate) shortly after birth to prevent clinical signs of anemia. Sow's milk has low quantities of iron, even when she is heavily supplemented prior to farrowing. As a result, newborn pigs will become anemic unless precautions are taken. This involves giving all piglets a single iron injection around 3 to 5 days of age. After the pigs are weaned off of milk, they will have access to natural iron in pasture and grain.

35. D: Infectious bovine rhinotracheitis. Infectious bovine rhinotracheitis is caused by a herpesvirus and results in clinical signs associated with upper respiratory infection such as anorexia, lethargy, sneezing, coughing, ptyalism, pyrexia, conjunctivitis, and a mucopurulent nasal discharge. Usually the symptoms resolve within a week providing there is no secondary bacterial pneumonia present. Immunization for this disease is recommended to prevent herd outbreaks.

36. C: Etomidate. Etomidate is an anesthetic induction agent that has a short duration of action with minimal effects on the cardiovascular system, which is ideal for a patient with a presumptive diagnosis of congestive heart failure. Etomidate does not increase or decrease heart rate or rhythm, respiratory rate or rhythm, or myocardial contractility, and has a long enough duration of action to allow for radiographs, an electrocardiogram, and a quick cardiac ultrasound.

37. D: Idiopathic megacolon. Feline idiopathic megacolon is a generalized dysfunction of colonic smooth muscle. Cats suffering from this syndrome collect large, impassable volumes of fecal matter in their colons that require medical intervention (e.g. rectal suppositories, enemas, manual extraction, laxatives) to eliminate. Some patients that are refractory to medical therapy may benefit from a colectomy.

38. E: Laryngeal hemiplegia. The most likely diagnosis is laryngeal hemiplegia. Laryngeal hemiplegia occurs as a result of damage (demyelination) to the

recurrent laryngeal nerve, which leads to atrophy of the cricoarytenoideus dorsalis muscle of the larynx. When this muscle is not properly innervated, it allows collapse of the left arytenoid cartilage into the airway during strenuous exercise, which produces the "roaring" noise during inspiration. Endoscopy provides a definitive diagnosis by allowing visualization of the partially or completely paralyzed left arytenoid cartilage. On physical exam, the prominent left muscular process of the arytenoid cartilage is a direct result of atrophy of the cricoarytenoideus dorsalis muscle.

39. A: Lead poisoning. Lead poisoning is the most likely diagnosis in this case based on the radiographic findings and recent history of paint ingestion (lead-based paint). Clinical signs of lead poisoning include vomiting, lethargy, inappetence, polyuria, diarrhea, and neurologic abnormalities. A presumptive diagnosis can be made with history and radiology; however, a definitive diagnosis is made by submitting a blood sample for a lead analysis. Treatment involves the chelator CaNa2EDTA to absorb lead from the body tissues for excretion. Enemas and emetics can also be used to remove residual lead in the GI tract. Surgery is sometimes required to remove larger lead pieces.

40. E: Seminoma. Seminomas are the most common testicular neoplasms of horses. They typically affect stallions that are 10 years of age and older. Seminomas are derived from spermatogenic cells and normally present as a progressive enlargement of one testicle over a course of weeks to several months; however, the condition can be bilateral as well. The scrotum is usually not painful or warm when palpated, as is commonly seen with inflammatory conditions. Ultrasonography usually reveals widespread heterogeneous echogenic areas with mixed hypoechoic and hyperechoic regions. Histopathology yields the definitive diagnosis. Treatment is surgical removal of the neoplastic testicle.

41. C: Phenylpropanolamine. Phenylpropanolamine is commonly used to manage urinary incontinence due to decreased urethral tone that is frequently seen in older spayed female dogs. This drug stimulates the sympathetic alpha-receptors in the smooth muscle of the urethra, which results in increased tone and subsequent decreased urine "dribbling."

42. C: Posterior synechia. Posterior synechia is an abnormal adhesion of the iris to the lens. In horses it is a common sequela of recurring or improperly managed anterior uveitis (the diagnosis in this case). A posterior synechia is significant in that it can give rise to a secondary cataract formation and subsequent blindness in horses. It is therefore imperative to treat this condition immediately with cycloplegics to dilate the pupil to prevent these adhesions as well as topical and systemic anti-inflammatories.

43. B: Necrotic laryngitis. Necrotic laryngitis is a bacterial infection of the oral cavity and larynx caused by the bacterium Fusobacterium necrophorum. It occurs as a result of damage/injury (ie, excessive bawling or coughing) to the mucous membranes that line and protects these structures. Once the injury takes place, opportunistic bacteria will invade and cause a devastating stomatitis, pharyngitis, and laryngitis, which result in painful eating and swallowing. Laryngeal swelling and inflammation will produce difficult inhalation. If caught early, the prognosis is good. If left untreated, the calf will quickly die of dehydration and widespread infection.

44. A: Clostridium perfringens. Clostridium perfringens can cause a fatal necrotizing hemorrhagic diarrhea in foals that are younger than 3 days old. Diagnosis is made by a fecal culture which will demonstrate large numbers of gram + rods, or via cytotoxin assay of fresh feces. Metronidazole is the antibiotic of choice for these patients, as well as intravenous fluid therapy and hospitalization.

45. E: Postparturient paresis. Milk fever, also known as hypocalcemic postparturient paresis, affects older, high-producing dairy cows within the first 72 hours of calving and occurs as a result of the rapid depletion of calcium during early lactation. Initially, the condition is marked by muscle tremors, weakness, and ataxia, which is the result of lowered calcium on muscle and nervous tissue. As the disease progresses, the cow becomes progressively weaker to the point where she can no longer stand. Her heart and respiratory muscles contract inefficiently, and, as a result, do not deliver blood or oxygen to the distant tissues. There is also GI stasis due to the effect of decreased calcium on smooth muscle contractions. The treatment of choice is intravenous calcium gluconate, which stabilizes serum calcium and dramatically reverses all the symptoms.

46. B: Urine. Bladder rupture with subsequent leakage of urine into the peritoneal cavity is a condition primarily of newborn foals. Bladder tears can occur during parturition as a result of strong uterine contractions on a full or moderately full fetal bladder. Often, affected foals urinate normally, especially when the bladder has only a small dorsal tear. Leakage into the peritoneal cavity will only then occur when the bladder is full, resulting in a slowly progressive abdominal distention. Fluid analysis will normally demonstrate creatinine and potassium levels that are higher than the blood chemistry values.

47. B: Amprolium. Amprolium is an effective drug for coccidial infections. The drug interferes with thiamine utilization and sexual reproduction of the protozoan parasite, thereby retarding the parasite's development.

48. B: Tetracycline. Eperythrozoonosis suis is a common finding in swine herds. It is spread when the blood of an infected pig comes in contact with the blood of a non-infected pig. This can occur with contaminated/reused syringes or surgical equipment, and biting insects such as lice. Infection is usually mild, and sometimes the pigs recover uneventfully without the need of antibiotics. The pigs that recover, however, become carriers and can develop severe clinical disease during times of stress. Clinical signs include weakness, anemia, staggering, inappetence, and lethargy. Insect control of lice and the addition of tetracycline to the feed of all pigs greatly reduce the incidence of the acute form of the disease, but do not prevent pigs from becoming chronic carriers.

49. D: Feline herpesvirus. Feline herpesvirus infection produces clinical signs associated with rhinotracheitis and ulcerative keratitis. It is transmitted by actively infected cats or asymptomatic carriers through grooming, airborne viruses, and/or fomites. Neonates that are infected usually die before 3 weeks of age. Older kittens between 6 to 12 weeks of age will demonstrate a severe rhinotracheitis with characteristic sneezing, nasal discharge, conjunctivitis, and fever, as well as an ulcerative keratitis that can be quite serious. In times of stress, the disease may recur in older cats that harbor a latent infection or an immunocompromised state (e.g., feline leukemia virus [FeLV]).

50. B: Methylene blue. Nitrates are found in high concentrations in fertilizer and certain plants (e.g., cereal grasses, sorghums) and result in toxicosis when consumed in large amounts by ruminants. Nitrates are converted by ruminal bacteria to ammonia and nitrite, the latter being highly toxic. Nitrite changes hemoglobin to methemoglobin by oxidizing the ferrous ion (Fe++) of hemoglobin. Methemoglobin has no oxygen-carrying properties, and so cannot deliver oxygen to the body tissues, thereby creating anoxia in the patient. Methylene blue converts methemoglobin back to hemoglobin by reducing the ferric ion (Fe+++) of methemoglobin back to the ferrous ion (Fe++) of hemoglobin.

How to Overcome Test Anxiety

Just the thought of taking a test is enough to make most people a little nervous. A test is an important event that can have a long-term impact on your future, so it's important to take it seriously and it's natural to feel anxious about performing well. But just because anxiety is normal, that doesn't mean that it's helpful in test taking, or that you should simply accept it as part of your life. Anxiety can have a variety of effects. These effects can be mild, like making you feel slightly nervous, or severe, like blocking your ability to focus or remember even a simple detail.

If you experience test anxiety—whether severe or mild—it's important to know how to beat it. To discover this, first you need to understand what causes test anxiety.

Causes of Test Anxiety

While we often think of anxiety as an uncontrollable emotional state, it can actually be caused by simple, practical things. One of the most common causes of test anxiety is that a person does not feel adequately prepared for their test. This feeling can be the result of many different issues such as poor study habits or lack of organization, but the most common culprit is time management. Starting to study too late, failing to organize your study time to cover all of the material, or being distracted while you study will mean that you're not well prepared for the test. This may lead to cramming the night before, which will cause you to be physically and mentally exhausted for the test. Poor time management also contributes to feelings of stress, fear, and hopelessness as you realize you are not well prepared but don't know what to do about it.

Other times, test anxiety is not related to your preparation for the test but comes from unresolved fear. This may be a past failure on a test, or poor performance on tests in general. It may come from comparing yourself to others who seem to be performing better or from the stress of living up to expectations. Anxiety may be driven by fears of the future—how failure on this test would affect your educational and career goals. These fears are often completely irrational, but they can still negatively impact your test performance.

Review Video: 3 Reasons You Have Test Anxiety
Visit mometrix.com/academy and enter code: 428468

Elements of Test Anxiety

As mentioned earlier, test anxiety is considered to be an emotional state, but it has physical and mental components as well. Sometimes you may not even realize that you are suffering from test anxiety until you notice the physical symptoms. These can include trembling hands, rapid heartbeat, sweating, nausea, and tense muscles. Extreme anxiety may lead to fainting or vomiting. Obviously, any of these symptoms can have a negative impact on testing. It is important to recognize them as soon as they begin to occur so that you can address the problem before it damages your performance.

Review Video: 3 Ways to Tell You Have Test Anxiety
Visit mometrix.com/academy and enter code: 927847

The mental components of test anxiety include trouble focusing and inability to remember learned information. During a test, your mind is on high alert, which can help you recall information and stay focused for an extended period of time. However, anxiety interferes with your mind's natural processes, causing you to blank out, even on the questions you know well. The strain of testing during anxiety makes it difficult to stay focused, especially on a test that may take several hours. Extreme anxiety can take a huge mental toll, making it difficult not only to recall test information but even to understand the test questions or pull your thoughts together.

Review Video: How Test Anxiety Affects Memory
Visit mometrix.com/academy and enter code: 609003

Effects of Test Anxiety

Test anxiety is like a disease—if left untreated, it will get progressively worse. Anxiety leads to poor performance, and this reinforces the feelings of fear and failure, which in turn lead to poor performances on subsequent tests. It can grow from a mild nervousness to a crippling condition. If allowed to progress, test anxiety can have a big impact on your schooling, and consequently on your future.

Test anxiety can spread to other parts of your life. Anxiety on tests can become anxiety in any stressful situation, and blanking on a test can turn into panicking

in a job situation. But fortunately, you don't have to let anxiety rule your testing and determine your grades. There are a number of relatively simple steps you can take to move past anxiety and function normally on a test and in the rest of life.

Review Video: How Test Anxiety Impacts Your Grades
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Physical Steps for Beating Test Anxiety

While test anxiety is a serious problem, the good news is that it can be overcome. It doesn't have to control your ability to think and remember information. While it may take time, you can begin taking steps today to beat anxiety.

Just as your first hint that you may be struggling with anxiety comes from the physical symptoms, the first step to treating it is also physical. Rest is crucial for having a clear, strong mind. If you are tired, it is much easier to give in to anxiety. But if you establish good sleep habits, your body and mind will be ready to perform optimally, without the strain of exhaustion. Additionally, sleeping well helps you to retain information better, so you're more likely to recall the answers when you see the test questions.

Getting good sleep means more than going to bed on time. It's important to allow your brain time to relax. Take study breaks from time to time so it doesn't get overworked, and don't study right before bed. Take time to rest your mind before trying to rest your body, or you may find it difficult to fall asleep.

Review Video: The Importance of Sleep for Your Brain
Visit mometrix.com/academy and enter code: 319338

Along with sleep, other aspects of physical health are important in preparing for a test. Good nutrition is vital for good brain function. Sugary foods and drinks may give a burst of energy but this burst is followed by a crash, both physically and emotionally. Instead, fuel your body with protein and vitamin-rich foods.

Also, drink plenty of water. Dehydration can lead to headaches and exhaustion, especially if your brain is already under stress from the rigors of the test. Particularly if your test is a long one, drink water during the breaks. And if possible, take an energy-boosting snack to eat between sections.

Review Video: How Diet Can Affect your Mood
Visit mometrix.com/academy and enter code: 624317

Along with sleep and diet, a third important part of physical health is exercise. Maintaining a steady workout schedule is helpful, but even taking 5-minute

study breaks to walk can help get your blood pumping faster and clear your head. Exercise also releases endorphins, which contribute to a positive feeling and can help combat test anxiety.

When you nurture your physical health, you are also contributing to your mental health. If your body is healthy, your mind is much more likely to be healthy as well. So take time to rest, nourish your body with healthy food and water, and get moving as much as possible. Taking these physical steps will make you stronger and more able to take the mental steps necessary to overcome test anxiety.

Review Video: **How to Stay Healthy and Prevent Test Anxiety**
Visit mometrix.com/academy and enter code: 877894

Mental Steps for Beating Test Anxiety

Working on the mental side of test anxiety can be more challenging, but as with the physical side, there are clear steps you can take to overcome it. As mentioned earlier, test anxiety often stems from lack of preparation, so the obvious solution is to prepare for the test. Effective studying may be the most important weapon you have for beating test anxiety, but you can and should employ several other mental tools to combat fear.

First, boost your confidence by reminding yourself of past success—tests or projects that you aced. If you're putting as much effort into preparing for this test as you did for those, there's no reason you should expect to fail here. Work hard to prepare; then trust your preparation.

Second, surround yourself with encouraging people. It can be helpful to find a study group, but be sure that the people you're around will encourage a positive attitude. If you spend time with others who are anxious or cynical, this will only contribute to your own anxiety. Look for others who are motivated to study hard from a desire to succeed, not from a fear of failure.

Third, reward yourself. A test is physically and mentally tiring, even without anxiety, and it can be helpful to have something to look forward to. Plan an activity following the test, regardless of the outcome, such as going to a movie or getting ice cream.

When you are taking the test, if you find yourself beginning to feel anxious, remind yourself that you know the material. Visualize successfully completing the test. Then take a few deep, relaxing breaths and return to it. Work through the questions carefully but with confidence, knowing that you are capable of succeeding.

Developing a healthy mental approach to test taking will also aid in other areas of life. Test anxiety affects more than just the actual test—it can be damaging to your mental health and even contribute to depression. It's important to beat test anxiety before it becomes a problem for more than testing.

Review Video: Test Anxiety and Depression
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Study Strategy

Being prepared for the test is necessary to combat anxiety, but what does being prepared look like? You may study for hours on end and still not feel prepared. What you need is a strategy for test prep. The next few pages outline our recommended steps to help you plan out and conquer the challenge of preparation.

Step 1: Scope Out the Test

Learn everything you can about the format (multiple choice, essay, etc.) and what will be on the test. Gather any study materials, course outlines, or sample exams that may be available. Not only will this help you to prepare, but knowing what to expect can help to alleviate test anxiety.

Step 2: Map Out the Material

Look through the textbook or study guide and make note of how many chapters or sections it has. Then divide these over the time you have. For example, if a book has 15 chapters and you have five days to study, you need to cover three chapters each day. Even better, if you have the time, leave an extra day at the end for overall review after you have gone through the material in depth.

If time is limited, you may need to prioritize the material. Look through it and make note of which sections you think you already have a good grasp on, and which need review. While you are studying, skim quickly through the familiar sections and take more time on the challenging parts. Write out your plan so you don't get lost as you go. Having a written plan also helps you feel more in control of the study, so anxiety is less likely to arise from feeling overwhelmed at the amount to cover. A sample plan may look like this:

- Day 1: Skim chapters 1–4, study chapter 5 (especially pages 31–33)
- Day 2: Study chapters 6–7, skim chapters 8–9
- Day 3: Skim chapter 10, study chapters 11–12 (especially pages 87–90)
- Day 4: Study chapters 13–15
- Day 5: Overall review (focus most on chapters 5, 6, and 12), take practice test

Step 3: Gather Your Tools

Decide what study method works best for you. Do you prefer to highlight in the book as you study and then go back over the highlighted portions? Or do you

type out notes of the important information? Or is it helpful to make flashcards that you can carry with you? Assemble the pens, index cards, highlighters, post-it notes, and any other materials you may need so you won't be distracted by getting up to find things while you study.

If you're having a hard time retaining the information or organizing your notes, experiment with different methods. For example, try color-coding by subject with colored pens, highlighters, or post-it notes. If you learn better by hearing, try recording yourself reading your notes so you can listen while in the car, working out, or simply sitting at your desk. Ask a friend to quiz you from your flashcards, or try teaching someone the material to solidify it in your mind.

Step 4: Create Your Environment

It's important to avoid distractions while you study. This includes both the obvious distractions like visitors and the subtle distractions like an uncomfortable chair (or a too-comfortable couch that makes you want to fall asleep). Set up the best study environment possible: good lighting and a comfortable work area. If background music helps you focus, you may want to turn it on, but otherwise keep the room quiet. If you are using a computer to take notes, be sure you don't have any other windows open, especially applications like social media, games, or anything else that could distract you. Silence your phone and turn off notifications. Be sure to keep water close by so you stay hydrated while you study (but avoid unhealthy drinks and snacks).

Also, take into account the best time of day to study. Are you freshest first thing in the morning? Try to set aside some time then to work through the material. Is your mind clearer in the afternoon or evening? Schedule your study session then. Another method is to study at the same time of day that you will take the test, so that your brain gets used to working on the material at that time and will be ready to focus at test time.

Step 5: Study!

Once you have done all the study preparation, it's time to settle into the actual studying. Sit down, take a few moments to settle your mind so you can focus, and begin to follow your study plan. Don't give in to distractions or let yourself procrastinate. This is your time to prepare so you'll be ready to fearlessly approach the test. Make the most of the time and stay focused.

Of course, you don't want to burn out. If you study too long you may find that you're not retaining the information very well. Take regular study breaks. For example, taking five minutes out of every hour to walk briskly, breathing deeply and swinging your arms, can help your mind stay fresh.

As you get to the end of each chapter or section, it's a good idea to do a quick review. Remind yourself of what you learned and work on any difficult parts. When you feel that you've mastered the material, move on to the next part. At the end of your study session, briefly skim through your notes again.

But while review is helpful, cramming last minute is NOT. If at all possible, work ahead so that you won't need to fit all your study into the last day. Cramming overloads your brain with more information than it can process and retain, and your tired mind may struggle to recall even previously learned information when it is overwhelmed with last-minute study. Also, the urgent nature of cramming and the stress placed on your brain contribute to anxiety. You'll be more likely to go to the test feeling unprepared and having trouble thinking clearly.

So don't cram, and don't stay up late before the test, even just to review your notes at a leisurely pace. Your brain needs rest more than it needs to go over the information again. In fact, plan to finish your studies by noon or early afternoon the day before the test. Give your brain the rest of the day to relax or focus on other things, and get a good night's sleep. Then you will be fresh for the test and better able to recall what you've studied.

Step 6: Take a practice test

Many courses offer sample tests, either online or in the study materials. This is an excellent resource to check whether you have mastered the material, as well as to prepare for the test format and environment.

Check the test format ahead of time: the number of questions, the type (multiple choice, free response, etc.), and the time limit. Then create a plan for working through them. For example, if you have 30 minutes to take a 60-question test, your limit is 30 seconds per question. Spend less time on the questions you know well so that you can take more time on the difficult ones.

If you have time to take several practice tests, take the first one open book, with no time limit. Work through the questions at your own pace and make sure you fully understand them. Gradually work up to taking a test under test conditions: sit at a desk with all study materials put away and set a timer. Pace yourself to make sure you finish the test with time to spare and go back to check your answers if you have time.

After each test, check your answers. On the questions you missed, be sure you understand why you missed them. Did you misread the question (tests can use tricky wording)? Did you forget the information? Or was it something you hadn't learned? Go back and study any shaky areas that the practice tests reveal.

Taking these tests not only helps with your grade, but also aids in combating test anxiety. If you're already used to the test conditions, you're less likely to worry about it, and working through tests until you're scoring well gives you a confidence boost. Go through the practice tests until you feel comfortable, and then you can go into the test knowing that you're ready for it.

Test Tips

On test day, you should be confident, knowing that you've prepared well and are ready to answer the questions. But aside from preparation, there are several test day strategies you can employ to maximize your performance.

First, as stated before, get a good night's sleep the night before the test (and for several nights before that, if possible). Go into the test with a fresh, alert mind rather than staying up late to study.

Try not to change too much about your normal routine on the day of the test. It's important to eat a nutritious breakfast, but if you normally don't eat breakfast at all, consider eating just a protein bar. If you're a coffee drinker, go ahead and have your normal coffee. Just make sure you time it so that the caffeine doesn't wear off right in the middle of your test. Avoid sugary beverages, and drink enough water to stay hydrated but not so much that you need a restroom break 10 minutes into the test. If your test isn't first thing in the morning, consider going for a walk or doing a light workout before the test to get your blood flowing.

Allow yourself enough time to get ready, and leave for the test with plenty of time to spare so you won't have the anxiety of scrambling to arrive in time. Another reason to be early is to select a good seat. It's helpful to sit away from doors and windows, which can be distracting. Find a good seat, get out your supplies, and settle your mind before the test begins.

When the test begins, start by going over the instructions carefully, even if you already know what to expect. Make sure you avoid any careless mistakes by following the directions.

Then begin working through the questions, pacing yourself as you've practiced. If you're not sure on an answer, don't spend too much time on it, and don't let it shake your confidence. Either skip it and come back later, or eliminate as many wrong answers as possible and guess among the remaining ones. Don't dwell on these questions as you continue—put them out of your mind and focus on what lies ahead.

Be sure to read all of the answer choices, even if you're sure the first one is the right answer. Sometimes you'll find a better one if you keep reading. But don't second-guess yourself if you do immediately know the answer. Your gut instinct is usually right. Don't let test anxiety rob you of the information you know.

If you have time at the end of the test (and if the test format allows), go back and review your answers. Be cautious about changing any, since your first instinct tends to be correct, but make sure you didn't misread any of the questions or accidentally mark the wrong answer choice. Look over any you skipped and make an educated guess.

At the end, leave the test feeling confident. You've done your best, so don't waste time worrying about your performance or wishing you could change anything. Instead, celebrate the successful completion of this test. And finally, use this test to learn how to deal with anxiety even better next time.

Review Video: 5 Tips to Beat Test Anxiety
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Important Qualification

Not all anxiety is created equal. If your test anxiety is causing major issues in your life beyond the classroom or testing center, or if you are experiencing troubling physical symptoms related to your anxiety, it may be a sign of a serious physiological or psychological condition. If this sounds like your situation, we strongly encourage you to seek professional help.

Thank You

We at Mometrix would like to extend our heartfelt thanks to you, our friend and patron, for allowing us to play a part in your journey. It is a privilege to serve people from all walks of life who are unified in their commitment to building the best future they can for themselves.

The preparation you devote to these important testing milestones may be the most valuable educational opportunity you have for making a real difference in your life. We encourage you to put your heart into it—that feeling of succeeding, overcoming, and yes, conquering will be well worth the hours you've invested.

We want to hear your story, your struggles and your successes, and if you see any opportunities for us to improve our materials so we can help others even more effectively in the future, please share that with us as well. **The team at Mometrix would be absolutely thrilled to hear from you!** So please, send us an email (support@mometrix.com) and let's stay in touch.

If you'd like some additional help, check out these other resources we offer for your exam:

http://MometrixFlashcards.com/NAVLE

Additional Bonus Material

Due to our efforts to try to keep this book to a manageable length, we've created a link that will give you access to all of your additional bonus material.

Please visit **https://www.mometrix.com/bonus948/navle** to access the information.

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